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Original Communications

ATELECTASIS AND BRONCHIECTASIS

AN EXPERIMENTAL STUDY CONCERNING THEIR RELATIONSHIP

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SINCE Heller,[24] in 1885, described a group of cases as "atelectatic bronchiectasis," a possible causative relationship between atelectasis and bronchiectasis has been considered more and more, especially during more recent times when postoperative massive pulmonary atelectasis became a well-known condition and was much studied. Heller[24] called "atelectatic bronchiectasis" cases which, in his opinion, had developed on the basis of persistent fetal atelectasis. More recently, the term "atelectatic bronchiectasis" has been used in a different sense. Since the introduction of bronchography (Sicard and Forestier) and the more intensive x-ray study of the chest, more and more evidence of postnatal atelectasis was found in a high percentage of cases afflicted with bronchiectasis. Investigations by pediatricians, especially those by Boyd[9] McNeil[39] Findlay[17] and Anspach,[4] have shown that atelectasis preceded bronchiectasis in many children. Thus, it seemed of particular importance to attempt a clarification of the causative role that atelectasis is supposed to play in the development of bronchiectasis. Another point was made of the fact that essentially similar mechanisms, namely bronchial obstruction, may lead to either atelectasis or bronchiectasis. Since Joerg, [28, 29] in 1832 and 1835, had described atelectasis as an entity that might develop in postnatal life, this condition has repeatedly stood in the foreground of interest. But in no period has it led to so many experimental investigations as in the last twenty years, after Pasteur,[44, 45] in various publications, had drawn renewed attention to postoperative atelectasis and Henderson[26] and Coryllos and Birnbaum[14] directed the attention of physicians and especially of surgeons to this

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condition, by contending that it was the first step in the development of postoperative bronchopneumonia and bronchiectasis, a step that could be avoided by preventive measures.

Traube,[63] in 1845, was the first to produce atelectasis experimentally. His work was elaborated by Lichtheim[37] (in 1879), who was the first to show experimental evidence for the absorption of the alveolar air into the blood in an obstructed lung. According to Bartels,[67] in 1860, Rudolph Virchow was the first who suggested this mechanism. Lichtheim's evidence is still valid today, although he was not able to give a satisfactory explanation for it. Such an explanation could not be given until modern physiology had furnished reliable instruments for measuring the tension of the blood gases. This became possible after the introduction of a microtonometer by Krogh[34] in 1909. Then it became known that tension of the gases in the arterial blood is in an approximate equilibrium with the gaseous tensions in the alveoli, but that this is not the case with the gases of the venous blood. This fact is explained, as Priestly and Haldane,[21] Henderson and Henderson[26] have stressed, by the fall of the oxygen tension by 60 mm. Hg during the flow of the blood through the systemic circulation, and the increase in the carbon-dioxide tension of 6 mm. Hg. Thus, the venous blood has a total gas tension of about 54 mm. Hg lower than the alveolar air. This difference in pressure must be regarded as the main cause of the absorption of the alveolar air in the lung, as soon as the free communication between the atmospheric air and the lung is interrupted.

Following his description of massive pulmonary atelectasis in cases of diphtheria and after operations, Pasteur considered reflex mechanisms rather than mechanical bronchial obstruction as the cause of atelectasis. Paralysis of the diaphragm in combination with active contraction of the lung was to be the cause of postoperative atelectasis. Scott,[55] Scrimger,[56] and others stressed bronchial spasms; other investigators thought more of posture and diaphragmatic paralysis or vascular constriction as essential causative factors.

These various theories were more or less supported by experimental studies. Sussig[41] Middeldorpf[41] et al. stopped ventilatory movements on one side by splitting the thoracic cage; de Rossi[41] Briscoe[10] 1920, Pasteur[45] 1890, Schroeder and Green,[64] divided the intercostal nerves; Elliot and Dingley[64] divided the phrenic nerve and made hemisection of the cord at the second and third segments, paralyzing the intercostal musculature and the diaphragm; Fontaine and Hermann[64] produced complete denervation of the lungs. All these experiments were not successful in so far as they were unable to bring about fairly regularly a condition comparable to massive pulmonary atelectasis, such as is observed in man following operations. This was possible only by mechanical obstruction of the air-conducting system of one lung. Such

experiments were performed by many investigators. Traube[63] used paper balls, to occlude bronchi, and Lichtheim[37] used laminaria rods; the balls swelled up and thereby remained in place, but not infrequently they expanded so much that the bronchus was ruptured. Heller[25] found this disadvantage so annoying that he employed rubber stoppers of appropriate size which were made to stick by burning them over a flame just prior to their introduction.

Nissen,[43] Middeldorpf,[41] and Strauss,[60] discarded the obstructive method from within because of its inherent difficulties and resorted to obstruction from without. In the dog, the conditions are somewhat more favorable than in the rabbit because a bronchoscope can be introduced and the bronchus can gradually be blocked while under direct inspection either with mastix or gum arabic (Band and Hall,[5] Adam and associates) or with tenacious mucoid material (Lee[35]) or with rubber sacks filled with iodine solution (Coryllos and Birnbaum[14]). Ecudero and Adams,[16] Tucker and Lee,[35] and Reinberg, Kopziosvkaja, and Zatkin[47] added important contributions to this subject. Another way was tried by Adams.[1] He placed a certain amount of silver nitrate (20 per cent) into a segment of a main bronchus, producing a fibrotic stricture. Moulonguet,[40] however, found that a solution concentrated enough to produce a stricture may also affect the bronchial and pulmonary arteries and may cause fatal hemorrhages and blood aspiration into the lung. Weaker solutions, however, were found to be unreliable in producing closure of the bronchus. Weinberg[68, 69] tried to bring about obstruction from within by introduction of sand, burs, teeth, peanuts, castor beans and inorganic material, such as nails.

In our experiments it was planned in the beginning to produce pulmonary atelectasis by obstruction from within in order to study the conditions which sometimes produced pulmonary atelectasis. We further wanted to study the sequence of events that may follow pulmonary atelectasis and their alleged relation to bronchiectasis. During the course of the experiments the original experimental plan was broadened, to include supplementary experiments which served the original purpose of clarifying the causative relationship between pulmonary atelectasis and bronchiectasis. Thus these eight series of experiments were finally conducted:

1. Obstruction from within the bronchus of one lung or one lobe.

2. The same, but with simultaneous induction of pneumothorax on the obstructed side and maintenance of the pneumothorax for the duration of the experiment.

3. Obstruction of the main bronchus of one lung from without by ligation directly below the bifurcation.

4. The same with simultaneous pneumothorax on the operated side.

5. Ligation of the pulmonary artery to one lung.

6. The same with subsequent or simultaneous infection.

7. Ligation of the main bronchus of one lung following intrabronchial deposition of infectious materal.

8. Partial obstruction of the main bronchus of one lung by ligation.

The parallel series of bronchial obstruction, with and without pneumothorax on the operated side, were made, because, in conformity with Andrus'[3] theory, it is often stated that a high negative intrapleural pressure in atelectasis is the force dilating the bronchi.

TECHNIQUE

1. Obstruction of the Bronchus From Within.—The obstruction of the main bronchus from within is connected with certain technical difficulties that each investigator experienced since the first attempt was made, so that practically each worker had to make his own choice between the advantages and disadvantages connected with different methods. The difficulties arise from these facts: the width of the bronchi in experimental animals is not a constant factor. The bronchi are functionally more or less dilated or constricted in conformity with the respiratory changes in their lumina. In rabbits the distance between carina and the branching-off of the upper lobe bronchus is very small, and the entrance into the main bronchus at the carina is somewhat narrower than the subsequent segment of the bronchus itself. Thus, the foreign body must have the thickness that permits it just to pass the carina under some pressure. On the other hand, the foreign body must not be so long that its posterior end would reach beyond the carina. A sudden obstruction of one main bronchus with a simultaneous partial obstruction of the contralateral main bronchus is more than can be tolerated by an animal. There is not as yet a standard method devised for this experimental purpose, as was shown by the multiplicity of techniques employed by former workers.

In our experiments, foreign bodies were introduced under local anesthesia through a transverse intercartilaginous tracheotomy. Prior to the tracheotomy, one or two silk threads were put through the wall of the exposed trachea by means of a curved needle. The incision was then made just above the threads. This afforded a simple means of holding the trachea in a fixed position during the following manipulations. Following the incision into the trachea a few drops of 4 per cent cocaine were dropped into its lumen and allowed to flow down toward the carina. It was possible to bring the foreign body into the left or right main bronchus by slightly stretching the end of the trachea held by the threads into a direction opposite to that of the bronchus to be obstructed. In order to eliminate cough which might eject the foreign body, the latter was wetted with a few drops of cocaine solution.

It is occasionally stated in the literature that rabbits cannot vomit and cough due to the weakness of their respiratory musculature. Taking the latter for granted, we experienced some disappointments at the start of our experiments when one of us tried to produce bronchial obstruction by dropping a lead-shot of appropriate size through the tracheotomy wound and letting it fall by its own weight into a bronchus. The rabbit was held in the vertical position, and maximal inspiration with dilatation of the bronchi was induced by having the rabbit breathe carbon-dioxide mixtures for several minutes. But even when the rabbits were kept for about fifteen minutes in a vertical position and the surface of the shots had been roughened, the rabbits were able to cough them up. Roentgenograms usually showed the shots later in the stomach. Even when two shots were connected by a short piece of wire, they were coughed up again, but could not pass the larynx, causing death by suffocation.

After this initial failure, a number of different foreign bodies were devised. Little glass tubes of appropriate size were fitted with wire barbs which protruded about 0.5 to 1 mm. over the surface, preventing backward movement of the foreign body by piercing the bronchial mucosa at such an attempt. Since, due to the functional variability of the width of the bronchus, even a plaster of Paris model could not indicate the exact size for a foreign body that would under all circumstances completely obstruct the bronchial lumen, thin pieces of dried sponge or slippery elm were fastened to the outside of the tiny glass tubes. They were expected to take up fluid, swell, and insure a complete obstruction. The glass tubes were filled with a barium paste in order to render them radiopaque. These foreign bodies were introduced with a copper wire of appropriate thickness and length. They were fastened to the wire just prior to their introduction by a drop of paraffin, so that the wire could easily be withdrawn after the foreign body was engaged in the bronchus. Later the glass tubing was replaced by rubber tubes; the swelling bodies at the outside were replaced by tiny sacs filled with a dextrose solution and yeast, and finally by an attachment of rubber sheets in parachute or umbrella-like formation. This last gadget, in our experience, showed the most feasible features, combining radiopacity with valvelike adaptability to the varying size of the bronchial lumen.

Ether anesthesia employed in the initial experiments was soon replaced by local anesthesia, because it was found that, if a successful obstruction of a main bronchus was achieved, the additional burden of general anesthesia could frequently not be tolerated. Quastel has shown that ether anesthesia depresses the respiratory center.

Strained respiration started as soon as the foreign body was introduced. Following the closure of the operative wound by suturing the

respective layers together, profound cyanosis and deep and labored respiration were observed, but within ten to fifteen minutes these usually began to subside, and half an hour to an hour following the operation the alarming dyspneic symptoms had usually disappeared. About twenty-four hours later the rabbits appeared approximately normal, moved about, and took food.

Either immediately or about one hour following the end of the operation, roentgenograms were taken to ascertain the location of the foreign body in the bronchus. Roentgenograms were repeated at weekly intervals or even more frequently if special questions required it. The first roentgenograms were always taken in lateral diameter in addition to a posterior-anterior direction; the former position was sometimes indispensable when the foreign body was projected into the x-ray shadow of the spine.

In a parallel series of rabbits pneumothorax was induced at the side of the lung whose bronchus was blocked. The induction of pneumothorax was done one day after the operation, in order to allow the rabbit to overcome the shock of the operation and adapt itself to the restriction of its respiratory surface. At the induction of pneumothorax 30 or 40 c.c. of air were injected and 20 to 25 c.c. were added every other day, depending on the intrapleural pressure. The amount of refills and their spacing were controlled by measuring the intrapleural pressure, and arranged so that the intrapleural pressure was never more negative than that in normal animals.

2. Obstruction of the Bronchus by Ligation.—Ligation of the bronchus was done in most instances on the right side. The ligation was made after intercostal thoracotomy under ether anesthesia and local anesthesia at the site of the ligation. We used ordinary cord of moderate thickness for ligating material. No attempt was made to isolate the bronchial arteries before ligation. The sudden deprivation of more than 50 per cent of respiratory surface, caused by the ligation of the right main bronchus, is a definite shock for the rabbit. Healthy rabbits stand the operation well. It took not more than forty to fifty minutes. Rabbits afflicted with some spontaneous disease, such as intestinal disturbances or marked coccidiosis, usually died soon following the operation without showing any evidence of local infection. By the time the thorax wound was closed, the respiratory rate had usually increased from the normal rate of about 60 per minute to 120, 150, or even 180. It was regarded as a prognostically bad sign when a rabbit was not able to increase its respiratory rate above 90 by the end of the operation. The deep cyanosis present at the end of the operation had usually subsided markedly by the end of the first half hour. The operation on the left side is technically easier, since the bronchus can more readily be exposed when the lung is pushed forward and downwards. Pressure breathing to avoid the effect of open pneumothorax was not used.

The absorption of the pneumothorax air was controlled roentgenologically, and it was found to be completed within a period of three to six days. It is not possible to state on roentgenologic evidence alone, whether or not the absorption of the pneumothorax gas is slowed by the atelectasis ensuing after bronchial obstruction. As control, a pneumothorax was established in twelve normal rabbits by the injection of 50 c.c. of air into the right pleural cavity. X-ray studies were made daily during the next three to six days. Since these rabbits were operated upon during these periods, the autoptic inspection of the exposed pleural cavity gave an additional control showing certain variations, even in normal rabbits.

Fig. 1.—Roentgenogram of rabbit 11, showing a dense shadow of the right lower lobe, eighteen hours after bronchial obstruction from within. The foreign body is visible in the seventh interspace. Clear space in the uppermost and lateral part of the field of the right lung shows that the right upper lobe is not atelectatic. This x-ray appearance remained unchanged throughout the survival period of twenty-nine days.

The operations were done under appropriate aseptic conditions. That we were successful in this respect is shown by the complete absence of local wound infections and empyemas in these rabbits.

3. Ligation of the Pulmonary Artery of One Lung.—The pulmonary artery was exposed under ether anesthesia by intercostal thoracotomy similar to the method used in bronchial ligation. A silk thread was used for ligation. The thorax was sutured in layers, so that the previ-

ous thoracic space was approximately re-established following the operation, as in the series mentioned above. In half the cases the right artery was ligated and in the other half the left. In the parallel series with infection, 0.01 mg. of bovine tubercle bacilli was injected intravenously one or several days following the operation.

Fig. 2.—Roentgenogram of the lung specimen of rabbit 11 that died twenty-nine days following bronchial obstruction. The foreign body at the entrance of the main bronchus has less than one-half the width of the distended bronchus distal to the obstructed area. Compare, also, the definite distension of the bronchi of the obstructed lung to the normal size of those of the contralateral lung.

4. Ligation of the Main Bronchus of One Lung Following Intrabronchial Deposition of Infectious Material.—The infectious material was obtained from saccular bronchiectases previously produced in other rabbits. This material was sometimes mixed with a loopful of bacterial growth isolated from a similar source. If there was no fresh material available, cultures were mixed with sterilized human sputum. The intratracheal injection of 0.3 to 0.5 c.c. of such material five to ten minutes prior to the operation for bronchial ligation resulted usually in death. The pulmonary reaction produced by the intratracheal injection of infectious material was apparently so extensive that the animals could not stand the additional respiratory restriction brought about by bronchial ligation. When the injection of infectious material was made into the exposed bronchus itself, just prior to the knotting of the ligature already in place, the procedure was better tolerated.

RESULTS

1. Bronchial Obstruction From Within, Without Simultaneous Pneumothorax.—Bronchial obstruction from within was produced in twenty-eight rabbits, in ten of which pneumothorax on the operated side was induced and maintained throughout the survival period. As the earliest sign of developing atelectasis, we found in the roentgenograms shift of the heart toward the side of the obstructed lung (Fig. 3). This occurred already about one hour after the operation (R. 24: 1 hour; R. 28: 50 minutes.) The shift of the heart was more easily and more early recognizable than a shadow, indicative of atelectatic lung tissue. The reason is this: If only one lobe becomes atelectatic, the shadow of that lobe, because of its small size, may be obscured by the shifted heart; the overinflated other lobes help to make the interpretation of the roentgenograms difficult. Definite shift of the heart toward the occluded side in combination with an elevated diaphragm on the same side was in some cases the only clear roentgenologic sign of atelectasis of single lobes in rabbits.

Fig. 3.

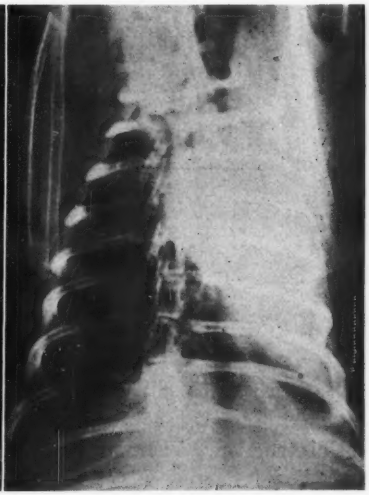


Fig. 4.

Fig. 3.—Roentgenogram of the thorax of rabbit 24, one hour after obstruction of the right lower lobe bronchus by a foreign body. Shift of the heart to the right side is the only indication of space made vacant in the right thoracic half.

Fig. 4.—Typical roentgenogram of pulmonary atelectasis following obstruction from within (rabbit 26). The shadow of the foreign body is seen within the fifth interspace. The heart and the right accessory lobe are shifted to the left side. The diaphragm is elevated by about two interspaces.

a. Early Changes Following Bronchial Obstruction.—Early death prior to achievement of atelectasis occurred in three rabbits in which the foreign body protruded too much into the trachea beyond the carina.

Complete atelectasis of one lung was found in a rabbit that died three hours after introduction of the foreign body. The occlusion of the main bronchus was so complete that formalin injected into the trachea under pressure of 40 cm. of water did not enter the obstructed lung. On the post-mortem roentgenogram (Fig. 5) the bronchial lumen distal to the foreign body was markedly constricted. Histologically there was

Fig. 5.—Roentgenogram of the lung specimen of rabbit 27. The rabbit had survived complete obstruction from within the left main bronchus, for three hours. At autopsy, formalin was injected into the trachea; no formalin penetrated beyond the foreign body. The roentgenogram taken after filling of the lung with formalin shows the reduced size of the atelectatic lung and the small slitlike appearance of its main bronchus compared to the wide bronchus of the right lower lobe.

almost complete atelectasis and constriction of the bronchi to the maximal degree permitted by the cartilaginous parts of their walls. The mucosa was markedly folded. This description might serve as an example of the animals that died during the first two days following bronchial obstruction. There was no difference whether or not a pneumothorax had been present on the obstructed side. Even in those animals that died after three hours, due to a spontaneous pneumothorax caused by piercing the bronchial wall, the atelectasis was more complete than could be attributed to pneumothorax alone. Pieces of the atelectatic lung placed in water did not float. It was also noted that in the rabbits that died after three and a half hours, the parahilar zone of the upper and lower lobes of the contralateral lung was atelectatic.

b. Effect of Short Periods of Bronchial Obstruction With Subsequent Spontaneous Removal of Foreign Body.—In five rabbits the bronchial obstruction had been maintained for periods ranging from seven to ten

days as evidenced by roentgenograms. After this time the foreign body had become dislodged. In all these animals which were kept alive from thirty to sixty days following the removal of the foreign body, the lung was found to be normal again. Only a few single mononuclear macrophages in some of the alveoli gave evidence of a former pathologic status in the lung. On the other hand, in one rabbit that had harbored the foreign body for a period of eighteen hours to six days and roentgenologically had shown complete atelectasis, it was seen that, after removal of the foreign body, the aeration of the lung did not take place as quickly as that of a fetal lung after birth. On the sixth day when the foreign body was found roentgenologically in the stomach (where it was found at autopsy thirty-two days later) the formerly obstructed lung still presented the picture of full atelectasis with a dense shadow, shift of the heart, an elevated diaphragm just as at the eighteenth hour period at which time the foreign body obstructed the bronchus.

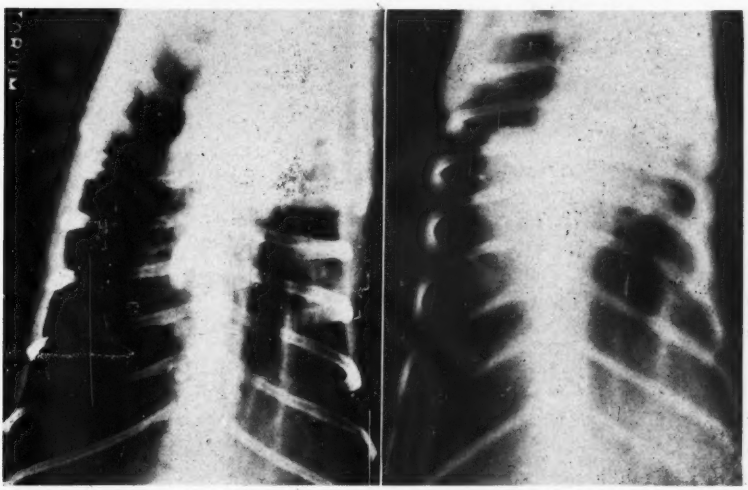


Fig. 6. Fig. 7.

Fig. 6.—Roentgenogram of the lung of rabbit 35, the main bronchus of which was obstructed from within fifteen days previously. Pneumothorax was maintained at the obstructed side.

Fig. 7.—Same as in Fig. 6, but three weeks later. A definite enlargement of the obstructed lower lobe is shown. This occurred in spite of maintenance of pneumothorax. The enlargement was due to development of saccular bronchiectases, as shown at autopsy.

c. Effect of Prolonged Bronchial Obstruction.—In all instances in which the foreign body had remained in the bronchus for more than a week, chronic inflammatory changes of the bronchial wall were seen at the site of the bronchial obstruction. The epithelium was shed in some areas and small ulcerations were present. In cases in which the foreign body had remained in place for several months, the bronchial

mucosa showed metaplasia of the columnar epithelium to stratified squamous epithelium in addition to polyblastic infiltration of the submucosa. Regularly, a strictly localized dilatation of the bronchial segment that had harbored the foreign body was observed.

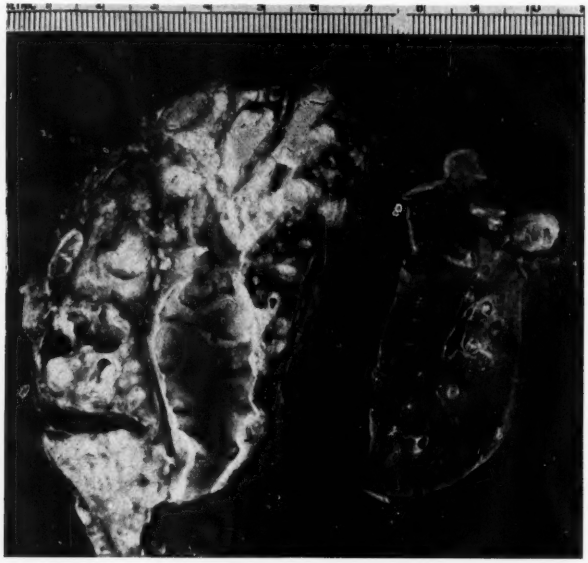


Fig. 8.—Gross appearance of the lower lobe of rabbit 36 that died twenty-nine days following bronchial obstruction from within, with simultaneous pneumothorax on the obstructed side. Compare the size of the saccular bronchi to those of the contralateral lung.

In this group are reported rabbits which have shown evidence of definite bronchial dilatation and saccular bronchiectasis, following bronchial obstruction from within and roentgenologic evidence of pulmonary atelectasis. These rabbits died spontaneously not earlier than two weeks after operation or were kept alive for two, three, four and almost six months. The presence and maintenance of a pneumothorax on the obstructed side did not essentially alter the final result; it did not prevent the formation of bronchiectases. In fact, it was found that the animals (55, 36) which showed the earliest evidence of definite bronchial dilatation, after fifteen days, and which exhibited the most extensive saccular bronchiectases (Figs. 8 to 10) belong to the series in which pneumothorax was maintained. Figs. 6 and 7 show that, in spite of a pneumothorax, the dense shadow of the lung grew up to considerable size during the period of three weeks. The autopsy showed that the extension of this shadow was produced by the immense dilatation of the bronchi with inflammatory exudate.

The most extensive bronchiectasis did not develop in those rabbits of this group which had the longest survival period but in those which died between the twenty-fifth and thirtieth postoperative day (Figs. 1 and 2). A typical example is rabbit 26, which exhibited bronchiectasis of the size of a finger in the obstructed left lung. A density over the left lung-field was seen eighteen hours after obstruction which remained unchanged until death (Fig. 4). At autopsy, about 3 c.c. of pus and fresh fibrinous pseudomembranes were found within the left pleural cavity. The fresh pleural exudate obviously was responsible for the final shift of the heart and the upper mediastinum to the right side that were seen on the post-mortem roentgenogram. Under the pleural surface of the left lung, round or oblong whitish areas were present, measuring from 1 to 5 mm. in diameter. The foreign body was found

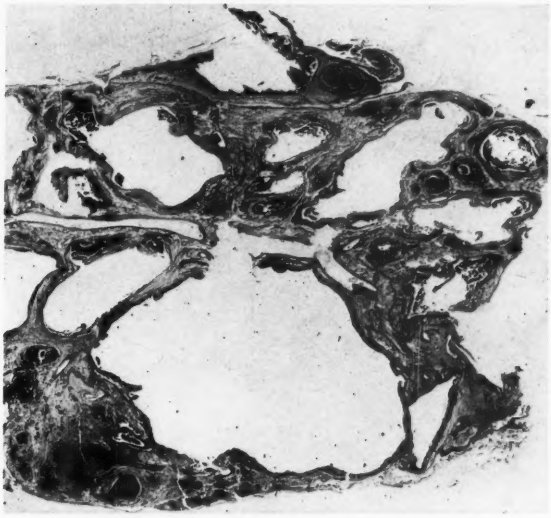


Fig. 9.—Section through the total lower lobe of the bronchiectatic lung of rabbit 36. The exudate from the saccular bronchiectasis was removed to show the relation of bronchiectasis to the remnants of the pulmonary parenchyma.

in the left main bronchus somewhat protruding beyond the carina into the trachea. After its removal large amounts of tenacious mucoid and puslike material poured forth which apparently was under a certain pressure. After fixation in formalin the further examination showed that the left main bronchus and its branches were maximally distended and changed to thin, saclike structures which reached to the pleural surface, accounting for the white subpleural foci, described above. The histologic examination showed that the bronchi were filled with a cellular exudate, composed mainly of polymorphonuclear leucocytes, which

had distended the bronchi to wide sacs and filled the smaller bronchi and bronchioli. The extent of the bronchial dilatation became especially apparent when their width was compared with that of their accompanying arteries which had retained their normal lumen. All layers of the bronchial walls were severely inflamed. The surface was ulcerated only in comparatively small areas.

Fig. 10.—Section through the bronchiectatic lung of rabbit 36 in higher magnification and with the purulent content present. See Figs. 8 and 9.

At the point where the cuboidal bronchiolar epithelium changes into the flat "alveolar epithelium," the polymorphonuclear intrabronchial exudate changed abruptly into an exudate of the character of that of a so-called desquamative pneumonia (Fig. 11). This exudate was composed mainly of round cells with large protoplasm, laden with nuclear debris, fragments of destroyed cells, especially of polymorphonuclear leucocytes (Fig. 12). There was a moderate number of multinucleated giant cells of an immense size, which, here and there, were arranged in

large clusters (Fig. 13). The marked phagocytosis of these cells in the alveolar exudate accounted for the disappearance of the polymorphonuclear leucocytes which became more infrequent with the increasing distance of an alveolus from its bronchiolus. In these lungs no normal air-containing parenchyma was present. The alveolar tissue surrounding the distended larger bronchi was collapsed and stretched in the direction of the expanded bronchus. At the transition of the bronchial ducts to alveolar tissue the latter was somewhat dilated and filled with exudate. In some areas small foci of fibrosis were observed. In the midst of newly formed fibrous tissue some slitlike openings were present lined by the cuboidal epithelium of bronchial ducts. In such areas of organization, the artery which accompanies a bronchial branch was narrowed or completely closed by organizing granulation tissue, composed of fibrous tissue and newly formed capillaries (Fig. 14).

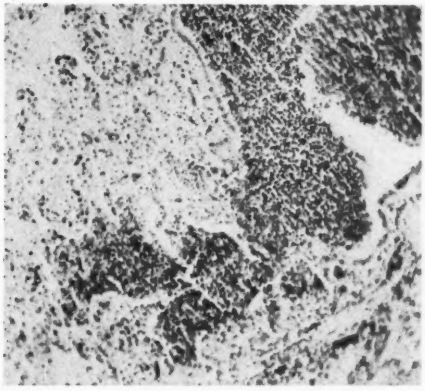


Fig. 11.—Border line between bronchial duct and alveoli in the lung of rabbit 11, twenty-nine days after bronchial obstruction from within. Note the change of the predominantly leucocytic exudate within the bronchus to desquamative-pneumonic exudate in the alveoli.

These findings are characteristic of the two parallel series in which bronchiectasis has developed following bronchial obstruction from within. Within the time limits of about four weeks to six months, the bronchiectases were, in general, the larger the shorter the survival time was. Ulceration and focal destruction of the bronchial wall were also more pronounced and more frequent in those animals in which the intrabronchial inflammation was more violent and the survival period shorter. The animals which survived more than two months showed only a moderate distention of the bronchi. The bronchiectases were more frequently cylindrical than saccular. The larger bronchi had a diameter not exceeding 5 to 8 mm. There was usually no purulent pleurisy. The pleural surface of these lungs had a livid, dusky-red color and was

subdivided into small fields which in their centers regularly showed gray-whitish slightly raised foci, 1 to 2 cm. in diameter. The longer the survival period the more grayish was the color of the subpleural ends of these dilated bronchioli instead of the pure white color of the more acute lesions. In histologic terms, in the older lesions, the bron-

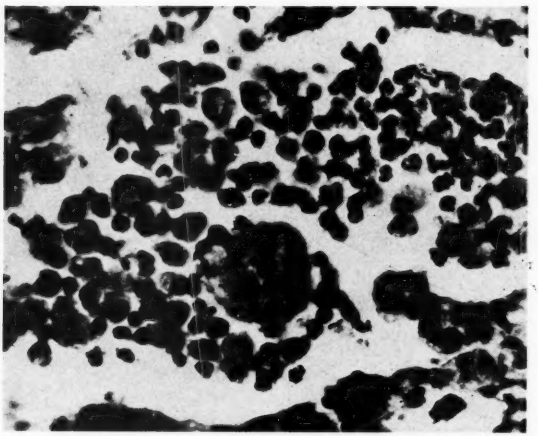


Fig. 12.—Type of alveolar exudate found in rabbit 11. The exudate is composed of rather large mononuclear phagocytes and multinucleated giant cells, only a few polymorphonuclear leucocytes being present.

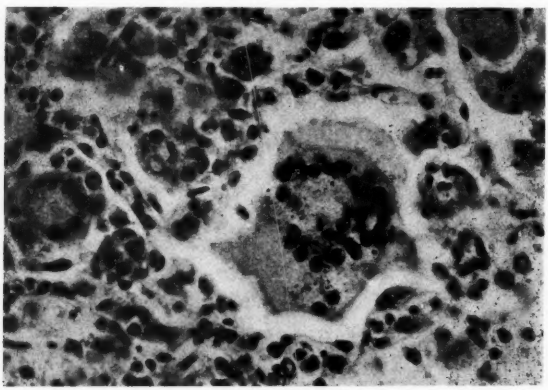


Fig. 13.—Multinucleated giant cells in the alveolar exudate of rabbit 11 that died twenty-nine days after bronchial obstruction from within.

chial exudate was less rich in polymorphonuclear leucocytes, more mucoid material, desquamated epithelial cells and mononuclear phagocytes being present. The inflammatory infiltration of the bronchial wall was correspondingly slighter, ulcerations being almost absent. When foci of fibrotic pulmonary parenchyma were observed, endar-

teritic processes were always seen, another indication of inflammatory irritation, in addition to simple atelectasis.

d. Prolonged Presence of Foreign Bodies in Bronchi Without Resulting Atelectasis and Bronchiectasis.—Three rabbits were observed which presented at autopsy a practically normal lung, in spite of the fact that they had harbored the foreign body in a larger bronchial branch

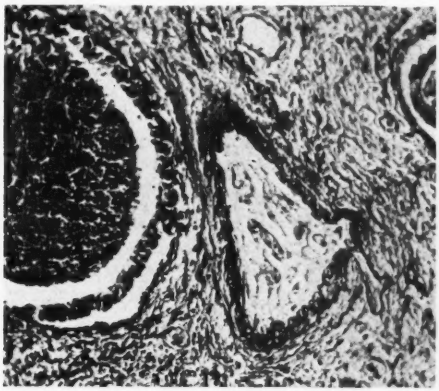


Fig. 14.—Area of carnification in the lung of rabbit 11. Obstruction of a branch of a pulmonary artery by formation of granulation tissue with newly formed capillaries within the arterial lumen. Note the purulent exudate in the lumen of the accompanying bronchus.

of the lower lobe for periods of thirty-two, forty-one, and eighty-nine days. The slight anatomical effect in the lungs of these rabbits was the more striking as the roentgenograms throughout the survival period indicated diminished aeration of the lung, shift of the heart to the side involved, elevation of the diaphragm and, at least during short periods, more or less distinct shadows around the foreign body. In the first two animals both lungs were found at autopsy to contain a normal amount of air. Filling of both lungs with formalin from the trachea under a pressure of about 40 cm. of water was easily possible, in spite of the presence of the foreign body in the anterior branch of the right lower lobe. In one rabbit some deeply retracted scars were found in the right lower lobe. There was no dilatation of the bronchi distal to the foreign body and they did not contain any secretion. The bronchial segment at the site of the foreign body was slightly dilated and showed a somewhat thickened epithelium. There was a slight chronic inflammation of the submucosa at this site and metaplasia of the normal columnar epithelium to stratified squamous epithelium. The pulmonary tissue showed, except for the few scars in one rabbit, no other pathologic changes in the portion supplied by the obstructed bronchus.

No atelectasis was found in the third rabbit (No. 19) which had harbored a foreign body for eighty-nine days. This lung was not inflated

intratracheally with formalin, but formalin was injected into the pulmonary artery under a pressure of 45 cm. of water. There was only a local dilatation of the bronchus at the site of the foreign body. Although the peripheral pulmonary portions were aerated, some mucoid material was present distal to the obstruction, but not enough to fill completely the bronchial lumen. Histologically, the bronchioli were found to be patent but not dilated. The appearance of the roentgenograms of this case suggested that the aeration of the obstructed part of the lung may have been impaired during part of the period of the experiment. Between the fiftieth and seventy-fifth postoperative day, the foreign body was surrounded by a moderately dense triangular shadow below the heart. The heart was dislodged toward this side. This, together with the presence of a residual leucocytic exudate in the obstructed bronchus may be taken as an indication that, at least off and on, a bronchitic inflammation had flared up distal to the bronchial obstruction. This might have caused complete obstruction of the bronchus and, at least temporary atelectasis of the parts of the lung, as suggested by some of the roentgenograms.

In summarizing the results obtained in these two series of experiments, we found that atelectasis following bronchial obstruction from within develops within a few hours.

As to the formation of bronchiectasis, we found that a period of about four weeks after bronchial obstruction was sufficient to produce extensive saccular bronchiectasis, involving not only one lobe but a whole lung. The bronchi in these cases were distended to the size of a finger of an adult. The largest bronchiectases we saw developed within the rather short period of about four weeks. Death of the rabbits was caused apparently by a contralateral bronchopneumonia, probably the result of intrabronchial aspiration of exudate from the bronchiectatic lung. When the rabbits lived longer, four or five months, and were then sacrificed, the bronchiectases were usually much smaller.

These experiments suggested that bronchial obstruction per se is not the decisive factor in the development of bronchiestasis. What seems to matter is the severity, the presence or absence of infection in the obstructed bronchus.

The second series of our experiments showed that pneumothorax cannot prevent the development of bronchiectasis. If the bronchial infection was severe enough, bronchiectasis developed in spite of pneumothorax. In fact, a few instances of the most severe and extensive bronchiectasis were obtained in such animals in which pneumothorax was maintained during the whole experimental period.

Two of the most striking results obtained are those in the rabbits in which foreign bodies had been present in the bronchus for about one to three weeks and in which, thereafter, complete restitutio ad integrum occurred roentgenologically, as well as anatomically. The other observa-

tion was that foreign bodies could be harbored for periods from one to three months in a bronchus producing nothing but a local dilatation at the site of their lodgement. But in these animals the bronchial obstruction was incomplete. The animal with the longest survival period in this group had given roentgenologic evidence of transient complete obstruction of the bronchus. Anatomically there were found signs of slight bronchitis, but for all practical purposes this inflammatory involvement had healed. This shows that the infection could only have been a mild one. This group also demonstrates that for the development of bronchiectasis, the severity of the bronchial inflammation is a most important factor. That it is, however, not the only causative factor is suggested by the observation that contralateral bronchopneumonia (i.e., without bronchial obstruction) never led to bronchiectasis.

2. Bronchial Obstruction by Ligation of the Main Bronchus of One Lung With and Without Simultaneous Pneumothorax at the Ligated Side.—The aim of these experiments was, (1) to establish, with more certainty than was possible in our previous experiments, that the main bronchus of an entire lung was completely closed and (2) to avoid with greater certainty intrabronchial infection.

Twenty-five rabbits were operated upon. In one series of thirteen rabbits, the initial operative pneumothorax was permitted to disappear by absorption; in the parallel series of twelve rabbits the initial pneumothorax was maintained throughout the survival period. The observations were extended over periods up to seven months.

a. Early Changes Following Bronchial Ligation.—Atelectasis developed within a period of not more than twenty minutes after the ligation. Immediately after ligation, the lung in such an instance was moderately collapsed, but its grayish-pink color, indicative of the presence of air, still prevailed. Twenty minutes later, the size of the lung had diminished definitely and the color had changed to a rather dusky red, such as is typical of complete or almost complete disappearance of the alveolar air from the lung.

Five rabbits died during the first day after operation. In all these rabbits the pneumothorax caused by the operation was still present. Death could invariably be explained in these rabbits by a weakened general condition, especially a marked, in one instance even an extremely severe coccidiosis of the liver. Special care was taken at autopsy to ascertain whether a pneumothorax of the contralateral side was to blame for the early death. This could be excluded in all animals. More or less marked pulmonary collapse was present between two and four hours after bronchial ligation. This observation corresponds to that made in the rabbits with bronchial obstruction from within which had died in a corresponding period of time. In the rabbits with bronchial ligation, the bronchi were found collapsed and con-

stricted within the atelectatic lungs to such a degree as was permitted by their anatomical structure, just as in the rabbits with bronchial obstruction from within.

b. Changes Following Prolonged Survival: aa. Uncomplicated Atelectasis.—The next group of ten rabbits is composed of animals which presented roentgenologically and anatomically, grossly and histologically, the picture of uncomplicated atelectasis. They all (except 2) belong, by chance, to the series in which pneumothorax was not maintained. They were sacrificed between the second and thirty-fourth day or died spontaneously between the nineteenth and fifty-first day after bronchial ligation. One rabbit was not killed, but the ligated lung was exposed and inspected during a second thoracotomy, 133 days after bronchial ligation. The ligating string was successfully removed during this second operation.

The roentgenograms showed during the whole experimental period typical changes: an evenly dense area corresponding to the atelectatic lung, marked shift of the heart to the ligated side, and elevation of the diaphragm. The latter, however, was frequently indistinct or absent. Instead, a clear space appeared below the dense shadow of the heart and that of the atelectatic lung, which in some instances reached across the shadow of the spine, indicating a direct continuation with the contralateral lung (Fig. 26). In other instances, this clear space below the shadow of the atelectatic lung was not so directly connected with the contralateral lung; its form was rather that of a triangle, the basis of which was laterally located. In several instances it was ascertained by bronchography that this space was a part of the contralateral lung. Fig. 31 illustrates this point. In all rabbits, however, the autopsy finally confirmed the x-ray observations (Figs. 15 to 20). In order to preserve the original position and the shape of the various lobes after death, the trachea was first exposed and tightly ligated. Then the abdominal cavity was opened, the intestines removed, and the diaphragm inspected. Opening of the pleural cavities and the removal of the lungs together with the trachea was the last step. This method of autopsy usually showed a much greater elevation of the diaphragm than could be expected from roentgenograms. It was not only the elevation of the diaphragm that contributed to the reduction of the thoracic space at the side of the atelectasis but also the reduction of the horizontal diameter of the diaphragmatic surface. The volume of the atelectatic lung was estimated to be a fifth or even less than that of a normally inflated lung (Fig. 15).

There was no pleural exudate in the pleural cavities that harbored the atelectatic lungs; neither were there massive pleural adhesions. Only a few tender threadlike adhesions were usually found. They sometimes formed a wide netlike meshwork between the pleural surfaces

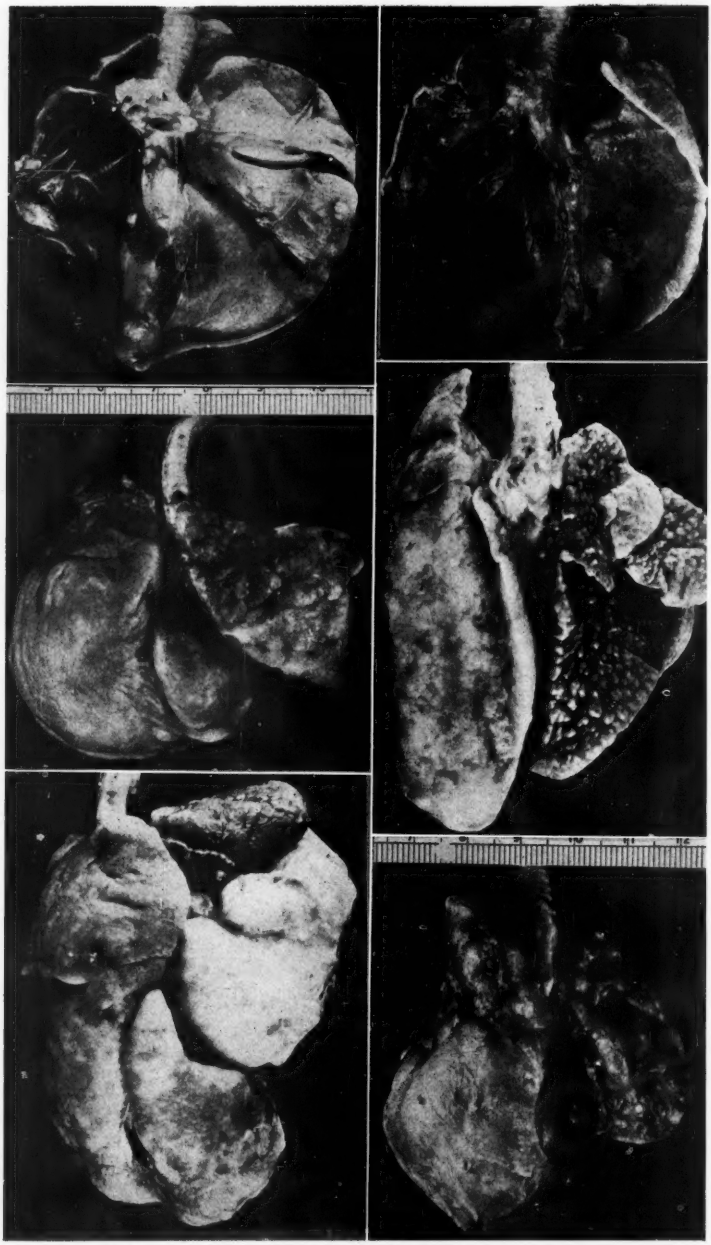
of the lung and the thoracic wall. The pericardial surface of the lung usually was more firmly adherent to the pericardium, obviously because of the operative manipulation during ligation.

In the rabbit that had been sacrificed two days following the bronchial ligation, the lung was completely atelectatic, although the pneumothorax was not yet completely absorbed. The lower posterior part of the left lung had been shifted into the right pleural cavity. A deep indentation of the spine on the posterior surface of the contralateral lung marked the borderline of the shifted part of the lung. In rabbits which had survived longer periods, this observation became more and more pronounced. This made the part of the shifted lobe of the contralateral lung appear as if a new lobe had been created. The displaced lobe in particular, but the entire contralateral lung as well, was overinflated (Fig. 24). The most extreme degree of such overinflation was observed in a rabbit in which the main bronchus of the left lung had been ligated. In this case the entire right accessory lobe and a large portion of the right lower and middle lobes had shifted into the contralateral side.

The appearance of the atelectatic lung was, aside from the markedly reduced volume, characterized by a smooth uniform surface that was livid, dusky-red with a slight brown hue. The collapsed lobules were barely recognizable as small fields of equal size. The atelectatic lung had a fleshlike consistency. On cross section, a cut-surface appeared which corresponded closely to the appearance of the surface. It was even, fleshlike, smooth, without granulations. The smaller bronchi were not recognizable, the largest lobar bronchi were slitlike and constricted. The markedly reduced size of the bronchi in the atelectatic lung was striking in comparison with those in the contralateral lung.

Histologically, the appearance of the alveolar tissue and that of the bronchi within the atelectatic lung corresponded to these gross observations. The alveoli were completely collapsed. There was neither edema nor cellular exudate in the alveoli. The bronchi were collapsed, the small ones completely, the larger ones as much as the architecture of their walls permitted; their mucosa was elevated in many folds, filling up thereby a good deal of the residual lumen. The bronchial epithelium still showed evidence of secretion, some goblet cells and some free bronchial secretion being present.

An interesting feature is presented by the rabbit which died fifty-one days after bronchial ligation, because of bronchopneumonia in the contralateral lung. The atelectatic lung had not been affected by this intercurrent infection. Figs. 4 and 26 show typical roentgenograms of simple atelectasis after bronchial ligation; Figs. 15 to 20, examples of the gross appearance of the atelectatic and contralateral lung; and Fig. 21, the histologic appearance of a lung that had been atelectatic



Figs. 15-20.—For legends see opposite page.

for fifty-one days. Those rabbits of this group in which, throughout the entire survival period, pneumothorax was maintained showed grossly and histologically identical changes, as far as the atelectatic lung is concerned. But there was, of course, no shift of the heart or the contralateral lung to the side of the ligation (compare Figs. 16 and 17). At autopsy, the diaphragm was bulging down into the abdominal cavity, and there was no indentation of the spine on the contralateral lung.

bb. Bronchial Ligation Complicated by Mild and Severe Infections.—A somewhat peculiar anatomical picture was presented in two rabbits which were sacrificed between ninety-five and one hundred days following ligation of the right main bronchus. In one of them, pneumothorax had been maintained throughout the survival period. Roentgenologically and at autopsy, both animals had presented the picture of uncomplicated atelectasis. The lungs were of the same small size and appearance as those with atelectasis. On cross section, made following fixation in formalin, it was however, observed that the most proximal segment of the large bronchi, distal to the ligature, was dilated to the size of a normal bronchus, or even a little more, and was empty. This dilatation continued for a certain distance into the atelectatic lobes, while the more distal bronchi were narrowed (Fig. 23). The site of the ligature on gross inspection and on probing was completely obstructed. Adams has described similar observations in his experiments. Since, such observations have been taken as evidence for the contention that bronchiectasis is produced by atelectasis, due to the pull on the outside of the bronchial tubes, a study of the site of the ligation in serial sections was undertaken. It was shown that at the

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Fig. 15.—Gross appearance of the lung of rabbit 109 which was sacrificed eight days after ligation of the right main bronchus. Note the small size and the comparatively dark color of the atelectatic right lung compared with that of the contralateral lung. The shift of a portion of the left lower lobe to the right thoracic side is indicated by a furrow produced by pressure of the spine.

Fig. 16.—Lung of rabbit 66, sacrificed ninety-six days following ligation of the right main bronchus. Pneumothorax was maintained throughout the survival period.

Fig. 17.—Lung of rabbit 67 that died ninety-eight days after bronchic ligation. A deep furrow on the posterior aspect of the left lower lobe, produced by pressure of the spine, marks the portion of that lobe that was shifted to the side of the ligated lung.

Fig. 18.—Lung of rabbit 94, posterior aspect. Sacrificed 141 days following ligation of right main bronchus. At autopsy, atelectasis and moderate cylindrical bronchiectasis. End of bronchioli appear as grayish subpleural slightly raised foci, 1 to 2 mm. in diameter. A furrow on the posterior aspect of the contralateral lung indicates to what degree this lung had been shifted to the side of the atelectatic lung.

Fig. 19.—Posterior aspect of lung and heart of rabbit 77, sacrificed 164 days following ligation of the right pulmonary artery. The right lung was shrunken to an almost quadrangular formation visible directly below the heart. A large portion of the left lower lobe, indicated by the deep indentation produced by the spine, had been shifted to the right side.

Fig. 20.—Gross appearance of the lung of rabbit 49 which was killed 123 days following ligation of the right main bronchus. Pneumothorax was maintained throughout the survival period. Nevertheless, a moderate degree of bronchiectasis developed. The bronchi were filled with mucoid material. The comparatively large size of the atelectatic lung indicates expansion which was due to bronchial dilatation. Pneumothorax prevented shift of the contralateral lung, absence of indentation of the spine.

side of the ligation a residual lumen of microscopical dimensions was present. It was lined, or better relined, by a simple layer of columnar epithelium, that in microscopic areas still was obviously attempting to bridge the last vestige of the ulcerated and chronically infiltrated base. The regenerating epithelium in these areas was of cuboidal shape.

This observation indicated that the mechanical trauma of the ligation had caused some damage to the bronchial wall and an inflammatory reaction, the exudate of which had contributed to a complete occlusion of the bronchus for some time, maybe weeks and months. Finally,

Fig. 21.—Section through the lower lobe of the right lung of rabbit 45, fifty-one days after ligation of the right main bronchus. Uncomplicated atelectasis with maximal constriction of the bronchi which contain very little secretions is shown. The bronchi are smaller than the arteries which accompany them.

however, the bronchial epithelium regenerated and re-established a new passage through the ligated areas. The pulmonary tissue, meanwhile, had become atelectatic. This property of the bronchial epithelium is analagous to that of the epithelium of the Fallopian tubes, which were found to establish again a passage after simple ligature, so that even pregnancy could occur. Following re-establishment of a microscopical lumen through the site of the ligature, air could again enter the lung.

The small size of the re-established lumen, however, must have had an effect comparable to that of a reducing valve. It would thus reduce considerably the pressure of the atmospheric air proximal to the ligature. According to the amount of air permitted to pass the site of the ligature, all transitional stages of reopening the bronchial tubes to full reinflation of the lung could be expected. If the amount of air readmitted was slightly greater than the amount of air absorbed, only the largest bronchi would be reopened; with increase in the amount of

Fig. 22.—Moderate bronchiectasis, lung of rabbit 94, sacrificed 141 days following ligation of the main bronchus of the right lung. Compare with the gross appearance of this lung, Fig. 18.

air, more and more small bronchi and finally alveoli would be opened. This is the reverse picture of what may be observed when lungs are becoming atelectatic: in each collapsing lobule the alveolar sacs collapse earlier than their bronchial duct.

In the instances which presented this picture, the pulmonary parenchyma remained atelectatic; the smaller bronchi were still narrower than normal, but contained mucoid matter with some shed-off epithelial cells and a rather large number of phagocytic mononuclear cells. Some bacteria of low-grade virulence had found access into the lumen of the

bronchial tree. The obstructing mucoid material within the small bronchi was another factor that prevented the reinflation of the atelectatic parenchyma.

cc. Moderate Bronchiectasis.—This group is composed of six rabbits that were sacrificed between the twenty-fifth and one hundred forty-first day following bronchial ligation. In these rabbits, the gross and

Fig. 23.—Lung of rabbit 67 that died ninety-eight days following bronchial ligation. The lobar bronchus of the right middle lobe is somewhat distended as a result of reopening of the bronchus at the site of the ligation as evidenced by serial sections. The alveolar tissue, for the most part, is still atelectatic. The smaller bronchi show almost normal width and contain little secretion; poor in cells.

histologic appearance was quite similar to that seen in rabbits with bronchial obstruction from within. At the surface of the lungs which, on the average, were of about twice the size of the lungs with uncomplicated atelectasis, the dilated bronchial ends were visible as slightly raised areas with a diameter of 1 to 2 mm. (Fig. 18). The alveoli were either atelectatic or were filled with an exudate characteristic of desquamative pneumonia. The exudate rarely was very cellular and neither was it massive so that the alveoli were small, definitely smaller than those of a normally inflated lung (Fig. 22). The bronchial and alveolar

exudates were regarded as the effect of a rather mild infection, that had either been present at the time of the ligation or had gained access through microscopic recanalization of the ligated portion of the bronchus. Serial sections of these areas suggested the latter possibility. Serial roentgenograms failed to show any evidence of a flare-up of an infection so that the possibility of the presence of incitants of low virulence within the bronchial tree at the time of the ligation cannot be excluded. There was nothing that indicated that the maintenance of a pneumothorax had influenced the process in any way.

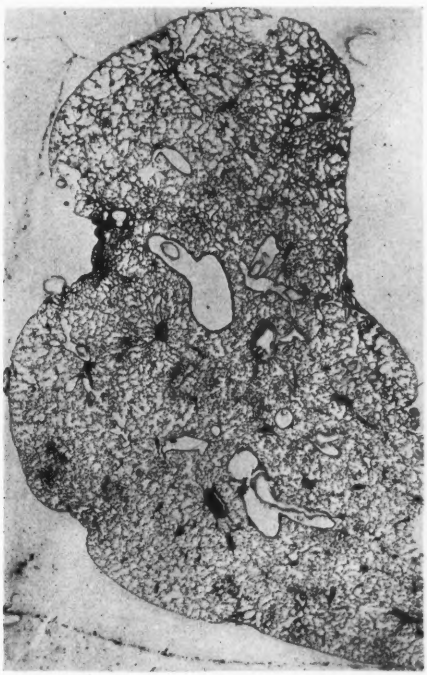

Fig. 24.—Horizontal section through the lower lobe of the left lung of rabbit 67, ninety-eight days following ligation of the right main bronchus. The deep indentation at the surface was produced by the spine, indicating to which degree the left lower lobe was shifted to the right thoracic side, into space made vacant by the atelectasis of the right lung. The entire left lung shows overinflation which is especially marked in the shifted portion of the lung.

dd. Complication With Severe Infection.—Rabbit 51 represents an example for the effect produced by a rather acute spontaneous and virulent infection of the bronchial tree. This rabbit was reoperated 133 days after bronchial ligation and presented, at that time, the picture of an uncomplicated atelectasis. At the second operation the ligating thread was removed. This had no demonstrable effect within the next weeks and months. As soon as the pneumothorax was ab-

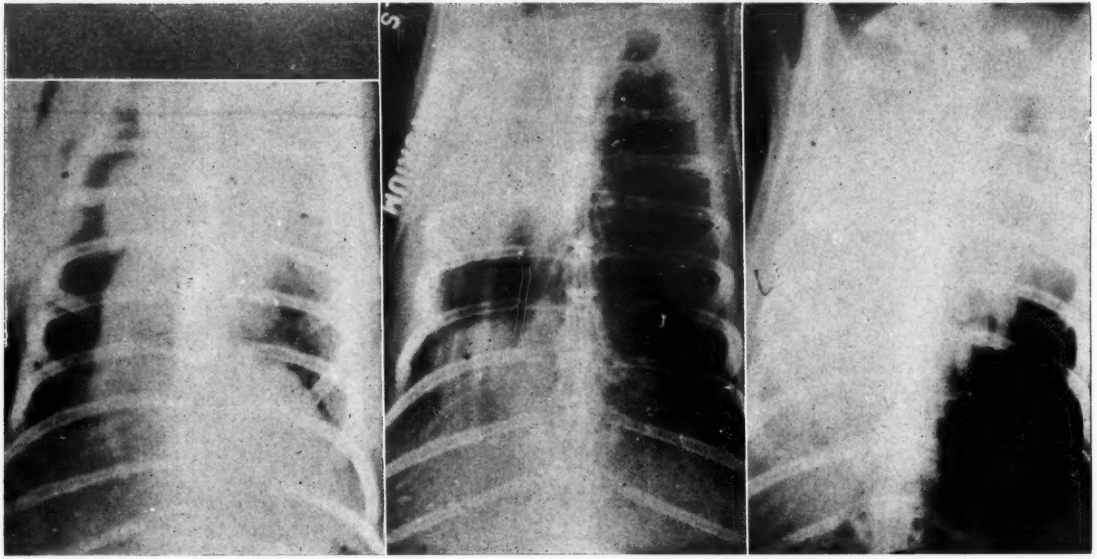

Fig. 25. Fig. 26. Fig. 27.

Fig. 25.—Roentgenogram of rabbit 51, one day following the second operation at which the ligature was removed that had been placed 133 days previously. Note the much reduced size of the atelectatic right lung which is surrounded by a pneumothorax.

Fig. 26.—Roentgenogram of rabbit 51, forty-six days after the second operation and 179 days after the first operation. The picture shows the same as that taken prior to the second operation; a small shadow of the atelectatic lung, shift of the heart and the contralateral lung to the right atelectatic side, and elevation of the right side of the diaphragm.

Fig. 27.—Roentgenogram of rabbit 51, eighty-six days after the second and 219 days after the first operation. Expansion of the formerly atelectatic lung is visible, its shadow reaching over to the contralateral side. The formerly shifted heart and contralateral lung have returned to their normal position.

sorbed, about six days following the operation, the roentgenograms showed the same picture as prior to the operation: shift of the heart and the contralateral lung to the side of the atelectatic lung (Figs. 25 and 26). In the days immediately following the operation, the atelectatic lung was seen as a distinct shadow against the clear pneumothorax space. There was no indication of any enlargement of the lung. Later roentgenograms showed that the lung remained unchanged until about

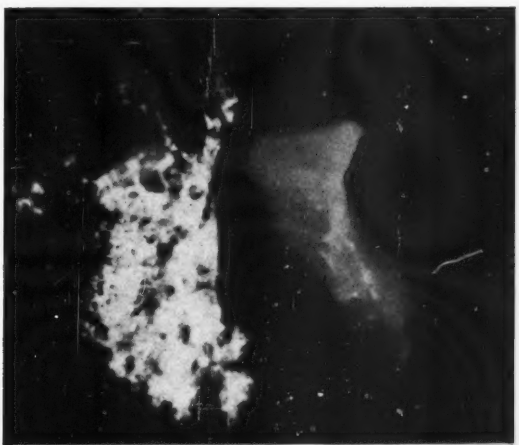

Fig. 28.

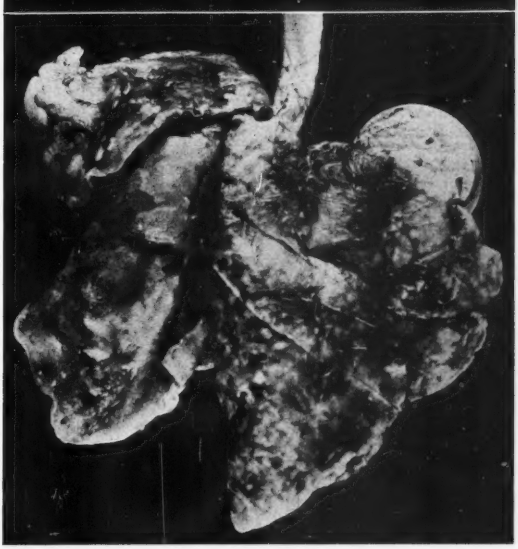

Fig. 29.

Fig. 28.—Roentgenogram of the lung of rabbit 51 following injection of 5 c.c. of iodized oil into the trachea. No oil penetrated beyond the ligation of the right main bronchus.

Fig. 29.—Lung specimen of rabbit 51, showing the marked enlargement that has occurred during the last four weeks prior to death.

four weeks prior to death, 219 days after the bronchial ligation and eighty-six days after the second operation. About three months following the second operation, roentgenograms showed for the first time a definite enlargement of the lung (Fig. 27), as indicated by the fact that the clear space, occupied by the displaced contralateral lung, disappeared and that the heart again became visible to the left from the sternum. After one week of increasing signs of dyspnea the rabbit was sacrificed. Autopsy revealed that the formerly ligated lung had expanded to more than the size of a fully inflated lung (Figs. 28 and 29). Its bronchi were now more than 1 cm. in diameter and, in some areas, could be seen below the pleural surface as pure white foci of more than 5 mm. in diameter. Large saclike bronchiectasis had developed in about one month after uncomplicated atelectasis had existed for several months, apparently caused by a severe infection.

ee. Total Fibrosis of the Lung Following Bronchial Ligation.—The only rabbits showing this lesion were killed three months following ligation of the left main bronchus. The postoperative course was uneventful; the roentgenogram showed atelectasis of the left lung with shift of the heart and the right lung to the left. The roentgenogram did not reveal the extraordinary shrinkage that was demonstrated by bronchography and at autopsy (Figs. 30 to 32). The left lung was so severely shrunken that its greatest diameter was not more than 2 cm. The grayish color exhibited also indicated a difference from the usual findings in an atelectatic lung. Cross section after fixation revealed that the entire parenchyma was replaced by fibrous tissue which surrounded the remnants of the bronchi. The bronchi were completely filled with a grayish-white exudate. The greatest diameter of the upper lobe bronchus was 2 mm., that of the left lower lobe, 4 mm. Histologically there was a microscopic opening under the ligature through which some bacteria may have passed into the bronchus, although it was closed by exudate and an almost completely fibrosed granulation tissue. The parenchyma was replaced by fibrotic tissue; the bronchial exudate contained surprisingly few leucocytes but much cellular debris, in part phagocytosed by large mononuclear cells and many multinucleated giant cells (Figs. 33 and 34). The lumen of the pulmonary arteries was almost completely closed by an organizing granulation tissue with newly formed capillaries inside the interior elastic membrane. This cirrhotic change of the lung had developed and reached its end result within three months. Infection, indicated also by the marked endarteritis of the pulmonary arteries, must be considered the cause rather than atelectasis per se. The actual dilatation of the remnants of the bronchi which occurred during this shrinkage of the lung was only very slight. Figs. 30 to 32 illustrate this case.

3. Ligation of the Pulmonary Artery, With and Without Infection.—This series of experiments was performed to decide the question

whether or not physical forces alone are able to produce bronchiectasis. It has been reported by several authors (Bruns and Sauerbruch,[11] Smirnoff,[57, 58] Krampf,[33] Schlaepfer[54]) that pulmonary atelectasis could be produced by ligation. We made a series of experiments on twelve rabbits which, after ligation of the pulmonary artery on one side, were left alive for a period up to five months. The lesions produced in the lungs of these rabbits, however, differ in principle from those produced by bronchial ligation. Soon following the arterial ligation hemorrhagic infiltrations and necroses developed, with the result that, if the animal survived the first postoperative period, organization of the necrotized alveolar tissue occurred with subsequent shrinkage of the lung. Thus, a condition is produced which resembles pulmonary atelectasis only superficially. The similarity of the sequellae of bronchial obstruction and ligation of the pulmonary artery lies in this: In either case the alveolar air disappears; in the former condition by absorption into the blood, in the latter by replacement with a hemorrhagic exudate. The latter infiltrates also the alveolar walls and fills the bronchi. In either case marked shrinkage of the lung follows. In atelectasis, induced by bronchial obstruction, the volume reduction of the lung follows immediately upon the bronchial ligation and remains stationary after the first few hours, unless complications occur. Following ligation of the pulmonary artery, the shrinkage of the lung is progressive for weeks, due to the organization of the hemorrhagic exudate and of the pulmonary parenchyma that has been necrotized by the intensive and extensive hemorrhagic infarction that marks the first stage of this process. For our question it is important that this extensive shrinkage of the lung (Fig. 19) takes place while the atmospheric air has free access to the bronchial tree. In the animals in which the shrinkage of the lung was not complicated by infection, no bronchiectasis of any appreciable size was obtained, although the animals were kept alive more than four and five months after the operation. Figs. 19 and 36 illustrate this statement.

In those animals, however, in which spontaneous infection intervened, or an artificial infection with a bovine strain of tubercle bacilli was produced, large bronchiectatic cavities were obtained.

It seems, therefore, that the conclusion is justified, that the mechanical forces operative when marked shrinkage of a lung occurs neither dilate bronchi nor produce bronchiestasis.

4. Ligation of the Main Bronchus of One Lung Following Intrabronchial Deposition of Infectious Material.—In this series, only five animals could be kept alive more than a few days. The results obtained are, however, significant, since they are complementary to the results obtained in the larger series reported above. After a survival period of four weeks, bronchiectases of a marked degree were observed as illus-

Fig. 30.

Fig. 31.

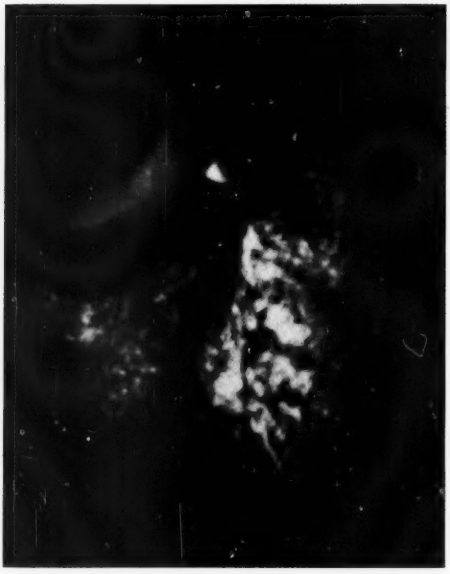

Fig. 32.

Fig. 30.—Posterior aspect of the lung of rabbit 126 which was sacrificed ninety days following ligation of the left main bronchus. The right accessory lobe, a large portion of the right lower lobe indicated by a deep furrow, and part of the right middle lobe had been shifted to the left side of the thorax into the space vacated by the shrinkage of the left lung.

Figs. 31 and 32.—Roentgenogram of the thorax of rabbit 126 following injection of iodized oil into the trachea. A small shadow in the upper left pulmonary field represents the fibrotic shrunken lung plus the shifted heart. The clear space below this shadow is occupied by shifted parts of the contralateral lung (Fig. 32). The x-ray of the specimen after death bears this out. No iodized oil has penetrated into the small shadow which represents the completely shrunken left lung. Compare with Fig. 30, showing the gross aspect of this lung.

trated in Fig. 35. Spontaneous death was caused by an extensive bronchopneumonia in the contralateral lung which was produced either by infectious material that was deposited in the bronchus just prior to ligation, or by bronchiectatic exudate that might have passed through the ligature during the later stage of the experiment. In the ligated lung, bronchiectasis and collapsed parenchyma were the dominant lesions. In the contralateral lung the main lesion was a bronchopneumonia with little, if any dilatation of the bronchi. One animal,

Fig. 33.—Site of ligation of the left main bronchus of rabbit 126, which was made ninety days prior to death. The bronchial wall is completely replaced by fibrous tissue. A residual lumen of microscopic dimensions, for the most part lined by regenerated epithelium, is seen in the upper part of the picture.

still alive three months after the ligation, showed roentgenologic evidence of slight expansion of the obstructed and infected lung during the first week and, subsequently, a progressive decrease of the pulmonary shadow. This suggested that a cirrhosis of the lung occurred, such as was observed in one rabbit with bronchial obstruction after a duration of three months, and such as was regularly observed in animals with ligation of the pulmonary artery.

5. Partial Ligation of the Main Bronchus of One Lung.—The results obtained in this series in which twelve rabbits were operated on are also reported as supplementary evidence for the conclusions based principally on the study of the series reported above. The partial ligation was done by the same technique as complete bronchial ligation, the ligation being made more loosely, so that a narrowing but no closure of the

Fig. 34.—Part of a cross section through the cirrhotic left lung of rabbit 126 that was sacrificed ninety days following ligation of the left main bronchus. The bronchi are filled with an exudate that shows many giant cells laden with leucocytic debris. The alveolar tissue is completely fibrotic except for a few slitlike openings which are lined by cuboidal epithelium.

bronchial lumen resulted. Five rabbits of this series have been killed, two of them in the second week, the other three between the seventy-second and one hundred seventy-fifth day following ligation. None of these five animals had bronchiestasis. The remainder of the animals are still alive, four and five months after the operation. Only results obtained by study of roentgenograms which were made at regular intervals during the survival period, are reported concerning the survivors. The partially obstructed lungs showed during the first one or two months a

reduced volume on roentgenograms, with shift of the heart of a moderate degree and elevation of the diaphragn at the operated side. The suggestion that this might be indicative of development of atelectasis in some areas in the lung was corroborated by autopsy observation on the rabbits sacrificed. Patchy atelectasis and compensatory overinflation of other areas of the lung were found in these instances. The overinflated areas exhibited histologically definite atrophy and disappearance of alveolar walls. The overinflation, therefore, had reached a degree in which it could be considered a true emphysema. In the animals which survived more than four and five months, emphysematous changes in the partially obstructed lung became more and more stable and predominant. They were characterized by expansion of the lung beyond the normal size, shift of the heart to the contralateral side, and lowering of the level of the diaphragm by one or two interspaces, as compared with the contralateral side. Pulmonary densities, present during the initial period of the experiment, disappeared later.

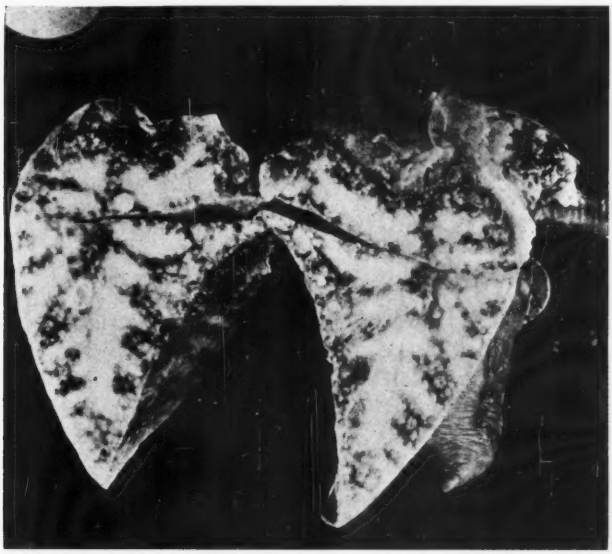

Fig. 35.—Cross section through the left lung of rabbit 136 showing the large distention of the bronchi by white purulent exudate. The rabbit died twenty-eight days after deposition of infectious material in the lumen of the left bronchus just prior to ligation of the bronchus.

CONCLUSIONS

A few definite conclusions seem justified. With regard to the development of pulmonary atelectasis, our experiments have only reaffirmed Traube's results in 1845, which showed that following bronchial obstruction complete atelectasis will occur within a period of two to

four hours. First evidence of developing atelectasis was found in the shift of the heart to the side of the obstructed lung in which the air was absorbed into the blood. This evidence was already available fifty to sixty minutes after bronchial obstruction, when the other roentgenologic signs of atelectasis, particularly increased density, were not as yet recognizable.

The experiments have shown that complete atelectasis may exist for a long time, up to several months, without complicating processes within the lung, provided that the atelectatic lung does not become infected.

Fig. 36.—Roentgenogram of the lung of rabbit 145 after injection of iodized oil into the trachea. The rabbit had survived ligation of the left pulmonary artery for forty-eight days. The left lung is completely shrunken; the main bronchi are filled with iodized oil but not dilated. Shift of the right accessory lobe to the left side and overinflation of the right lung has compensated for the shrinkage of the left lung and filled the space made vacant.

Even though almost complete obstruction of a bronchus from within is likely to favor infection of the lung by impairing the self-cleansing process, one group of experiments has shown that obstructing foreign bodies can remain for many weeks within bronchi without producing any disturbances, save a strictly local dilatation of the bronchial tube at the site of their lodgment. The most probable explanation for this unexpected observation is that the dilatation of the bronchus at the bed of the foreign body soon permits the air to pass through the obstruction. On the other hand, Van Allen's so-called collateral respiration may contribute to bring about the aeration of the pulmonary parenchyma distal to the bronchial obstruction by way of Kohn's pores in the alveolar walls. The elimination of bronchial secretions distal from the obstructed area suggests, however, that the first mechanism plays the more important role.

It has also been shown that pulmonary atelectasis up to ten days' duration, produced by bronchial obstruction from within may completely disappear after removal of the obstructing foreign body, without leaving any trace of the former involvement of the lung.

It has definitely been shown that in pulmonary atelectasis, produced by bronchial obstruction from within or from without, the bronchi of all sizes distal to the obstruction participate in the pulmonary collapse, becoming as much constricted as is permitted by the structure of their walls; the reason for their collapse is apparently the same as that for the parenchymal collapse.

The experiments have also shown how the potential space made available by the shrinkage of the atelectatic lung is filled. All neighboring structures and organs which are directly or indirectly under atmospheric pressure, tend to occupy the space formerly filled by the expanded lung.

Pulmonary atelectasis should be distinguished strictly from other processes which lead to a reduced volume of the lung that may have similar or identical roentgenologic appearances. The sequelae of ligation of the pulmonary artery are roentgenologically indistinguishable from obstructive atelectasis. Pulmonary artery ligation produces first pulmonary edema and hemorrhagic infarction, then organizing processes which result in fibrotic changes of parts of the pulmonary parenchyma, and not infrequently in fibrotic shrinkage of the whole lung. This may develop without complicating infection. This course of events is, however, so different from that of uncomplicated atelectasis that a sharp distinction should be made. In the course of pulmonary atelectasis, complications may occur which lead to the same end stages, namely, complete fibrosis and maximal shrinkage of the lung. But when this occurs in obstructive atelectasis, it is not a consequence of atelectasis per se, but it is due to complicating infection.

The experiments have also proved that localized pressure exerted on the surface of the lung may lead to strictly localized atelectasis by compression. This has been observed in the furrowlike indentation which was produced by the spine on the posterior surface of the contralateral lung that was displaced into the side of the atelectasis. Although the shifted portion of the lung was overinflated, the subpleural area below the furrow was collapsed or atelectatic. Similar observations were made when animals died during an early period after bronchial ligation on the right side. In these cases the parahilar areas of the left lower and upper lobes, which were exposed to the weight of the heart during the operation, were found to be atelectatic. The interalveolar openings (Kohn's pores), which make possible Van Allen's collateral respiration, are considered to be responsible, also, for disappearance of air from pulmonary tissue which is exposed to a localized pressure for some time.

With regard to the formation of bronchiectasis, these conclusions can be drawn: The mechanical forces (high negative intrapleural pres-

sure) operative, when following bronchial occlusion the alveolar air is absorbed, do not tend to dilate, but rather to constrict the bronchi. Uncomplicated pulmonary atelectasis per se can, therefore, not be a cause of bronchiectasis. Even if the access of air to the bronchial tree was not prevented (by ligation or obstruction from within), namely in ligation of the pulmonary artery, the increased negative intrapleural pressure did not produce dilatation of the bronchi, unless the process was complicated by accidental or intentional infection.

On the other hand, the experiments have shown that an infection may produce, within a period of from three to four weeks, most extensive saccular bronchiectases within a formerly atelectatic lung. They have further shown that the increased negative intrapleural pressure at the side of an atelectatic lung is an irrelevant factor in the pathogenesis of bronchiectasis, since bronchiectases of the same intensity developed within the same time, whether or not highly negative intrapleural pressures were prevented by induction and maintenance of pneumothorax.

The arrangement of the collapsed alveolar tissue which is stretched in the direction parallel to the circumference of the dilated bronchi, among other factors, suggested strongly that the pressure of the inflammatory exudate within the bronchi, which cannot be evacuated because of bronchial obstruction, is probably an important factor in dilating bronchi whose walls have become weakened by inflammatory involvement.

That kind of infection is most likely to produce bronchiectasis that is virulent enough to cause a purulent inflammation within the bronchi, but that is not virulent enough to produce the same kind of exudate within the alveoli but only a desquamative pneumonia. More virulent infections apparently led to death before bronchiectatic changes could develop. Bronchial and pulmonary infections never resulted in bronchiectasis when the bronchial lumina were not severely obstructed. An intrabronchial aspiration of bronchiectatic exudate into the contralateral lung caused bronchopneumonia and not bronchiectases. It must, therefore, be concluded that the association of two pathogenic factors is necessary for the development of bronchiectases: infection and impairment of bronchial drainage.

COMMENT

We wish to discuss whether the results obtained in our experiments have any bearing on the understanding of the pathogenesis of bronchiectasis in man. The first point that became clear in our experiments is that, in lungs with uncomplicated atelectasis, the bronchi are not dilated but constricted as much as their anatomical structure permits. This is the same in human pathology, in spite of claims to the contrary which are usually based either on purely theoretical considerations, or on the fact that in cases of bronchiectasis there is not infrequently a history

of pulmonary atelectasis. The evidence we wish to adduce for our contention that in human pathology the bronchi are also closed or constricted was found in a critical survey of the literature. The cases were reported without bias and without thought that they may have any bearing on a theory concerned with the causative relationship between atelectasis and bronchiectasis. The first case of this kind, a shrunken atelectatic pulmonary lobe with maximally constricted bronchi, was reported by Reynaud[48] in 1835; Biermer,[8] in 1860, mentioned that he had made several simliar observations. Ratjen,[46] in 1867, published a case under the title of congenital defect of the lung. In a man of 49 years he found a completely atelectatic lung. The bronchus was closed for a distance of 1.5 inches below the carina, but patent, although definitely constricted, distal to the obliteration and it contained a moderate amount of mucus. Ratjen's interpretation as a congential lesion cannot be accepted, for he described that the atelectatic lung showed the usual anthracotic pigmentation. In modern times, such cases are also reported. Thus, Loeschke,[38] in 1928, reported several cases of kyphoscoliosis in which atelectasis had occurred, due to kinking of the main bronchus of a middle lobe. Descriptions and pictures of these cases are evidence that the bronchi distal to the kinked area were as much constricted as their structure permitted. The same is true of cases which are reported by Roessle[50] in 1935, in connection with his anatomical study of epituberculosis. In these cases, closure of a lobar bronchus was caused by circumscribed tuberculous granulomata, obstructing bronchial lumina. In America, similar cases have been reported quite recently by Terplan[51, 62] and his associates.

The counterpart of sudden, complete obstruction of a normal main bronchus leading to a normal lung does not exist in human pathology. When a lobar bronchus becomes occluded, it is usually by a slow process, such as intrabronchial or peribronchial tumor growth, by pressure of lymph nodes or by intrabronchial tuberculosis. In such cases, the status of complete closure is preceded by a period of increasing stenosis, during which any complicating infection is prevented from healing by the impairment of bronchial drainage. In cases in which bronchial occlusion occurs fairly suddenly, such as in massive postoperative collapse, the lung or the bronchi are not normal, otherwise there would be no secretions to bring about bronchial occlusion. In either case, bronchial occlusion is not followed by simple atelectasis, but by atelectasis plus inflammatory changes, that may or may not lead to irreversible processes. This is amply confirmed by autoptic findings. In chronic obstructive processes, high-grade stenosis is infinitely more frequent than complete occlusion, as evidenced by bronchoscopic and necroptic observations. In sudden occlusions, the clinical features of fever and leucocytosis are always present, indicating clearly the inflammatory complications.

The aspiration of a foreign body hardly ever causes immediate bronchial closure. Occlusion usually develops in the train of the inflammatory reaction that is caused by the foreign body and the infection that is carried along. In other words, in these cases, too, complete occlusion is preceded by stenosis.

These considerations explain why uncomplicated chronic atelectasis is an extremely rare occurrence in human pathology, if, indeed, it ever occurs.

The generally accepted roentgenologic evidence of atelectasis, such as ground-glass shadow, thoracic retraction, mediastinal shift, and elevation of diaphragm, is not pathognomonic for simple atelectasis; it is diagnostic of the decreased volume of one lung. This may be caused by simple or complicated atelectasis or by any process that, without occluding a lobar bronchus, has produced fibrotic shrinkage of a sufficiently large portion of one lung. (See the results of pulmonary artery ligation.)

The second main point brought out by our experiments on rabbits is also supported by experiences in human pathology. This is the fact that space "made vacant" in one side of the thorax by shrinkage of a lung, either by atelectasis or by fibrosis, is not filled by overdistention of the bronchi, but by overinflation or emphysema of the contralateral lung and shift of other neighbouring organs. Ratjen[46] described that the space "made vacant" by the shrunken left lung was occupied by the overinflated right lung, which, by the way, showed on its posterior surface a deep furrow produced by the pressure of the spine, a mark indicating to which degree the contralateral lung had been shifted to the other side. Loeschke[38] found in his cases of kyphoscoliosis, emphysematous changes in the contralateral lung. In the majority of cases of shrunken lungs in human beings, the involvement does not extend throughout one whole lung. Therefore, both the uninvolved parts of the isolateral lung and the contralateral lung will, by overinflation, help to fill the space formerly occupied by the fully inflated lung.

A comparison of the experimental observations with the changes seen in human pathology makes it the more probable that uncomplicated atelectasis per se has no bearing whatever on the development of bronchiectasis. There is, however, another relationship of these two conditions. Both may be produced by the same cause. Atelectasis in children and the other primary diseases which are found frequently in the history of bronchiectasis are caused almost without exception by inflammatory conditions in the lung. Bronchial exudate and inflammatory swelling of the bronchial mucosa may close the bronchial lumen and cause atelectasis within a short time. The observation of Warner and Graham,[61] 1933, that bronchiectasis in man may develop in such a short time that the more chronic process of fibrosis cannot account for it concurs fully with our results obtained in animal experiments. We have shown experimentally that bronchiectasis never develops without infection; this

factor is so dominant that it made no difference in our experiments whether or not we eliminated highly negative intrapleural pressure by artificial pneumothorax.

As to the question what kind of infection is to blame, the specific bacteriologic investigations of bronchiectasis, as conducted by Smith,[59] Grey[29] et al. have not led to any definite results. Neither did the bacteriologic studies we conducted during our experimental work. Erb,[15] in 1933, certainly was right in saying "the bacteriology of bronchiectasis is the bacteriology not of the chronic bronchiectatic lung, but of the bronchopneumonia or other acute respiratory diseases which mark the onset of symptoms." We wish to stress two points in this respect. First, the infection must be of the kind that the wall of the bronchi is severely involved and infiltrated by inflammatory exudate to a degree that muscular constriction is greatly impaired, either by mechanical destruction or by paralysis of their nervous apparatus. This will impair the elimination of the bronchial secretions and the inflammatory exudate by the normal mechanism. They will accumulate and exert increasing pressure on the weakened wall of the bronchi, in the same way as any inflammatory exudate produces definite pressure on the surrounding tissue. It is well in accord with this consideration that usually the lower lobes are involved by bronchiectasis. Biermer,[8] in 1860, was probably the first to note the insensibility of bronchiectatics to accumulation of large amounts of secretion in their bronchi, and he compared it with the hypersensitivity of persons suffering from whooping cough. In recent years, Sunder-Plassmann[61] in his specific histologic studies on the nervous apparatus of the bronchial wall, found in lobes, removed for bronchiectasis, that the local nervous apparatus was destroyed by inflammatory and scarring products. Lemoine[36] has compiled cases from the literature in which thrombosis of the bronchial arteries was observed together with bronchiectasis and reported experiments in which bronchiectasis was attained after closure of bronchial arteries. The additional factor that supported the infection in these cases was the nutritional impairment of the musculature of the bronchial walls. This factor might well account for the insufficient activity of the bronchial musculature in removing exudate and secretions from the bronchial tubes.

Second, in our experiments, an inflammation seemed to be most likely to produce bronchiectasis, that was of a virulence able to produce a purulent exudate within the bronchi and a desquamative pneumonic exudate within the respective alveolar parenchyma. However, the identical type of infection per se did not cause bronchiectasis if it was not complicated either by complete, or incomplete bronchial obstruction. This is convincingly shown in those of our rabbits that had bronchiectasis and contralateral bronchopneumonia. On the contralateral side there was no impairment of bronchial drainage, hence no bronchial dilatation.

We see no need to resort to other explanations for the formation of bronchiectasis than those discussed above. The theory of Sauerbruch[52] that most bronchiestases, more than 80 per cent, are congenital is not supported by valid evidence, and pathologists with great experience have not accepted this view (Muller,[42] Hart,[23] Erb,[15] Hueter,[27] Robinson[49]), due to the failure of finding congenital bronchiectasis in infants, save as rare exceptions. In recent years this theory has received some support by finding a peculiar triad that occurs sometimes: situs inversus, bronchiectasis, and sinusitis. Such cases are reported by Kautsky,[31] Adams and Churchill,[2] Cole and Nalles,[12] and others. They are exceptions, after all. It might be possible to consider a congenital weakness of the structure of the bronchial wall as a predisposing factor in persons who become afflicted with bronchiectasis. More evidence for such a contention, however, has to be brought forth before such a theory can be accepted.

SUMMARY

1. Pulmonary atelectasis, as produced by obstruction from within or by bronchial ligation in rabbits, may exist for periods up to several months without any complications in the affected lung, if no infection intervenes.

2. Uncomplicated pulmonary atelectasis per se does not tend to dilate the bronchi. The same force that brings about atelectasis also causes collapse and constriction of the bronchi.

3. Increase in the negative intrapleural pressure on the side of the atelectatic lung does not dilate the bronchi of the atelectatic lung.

4. The increased negative intrapleural pressure on the side of the atelectatic lung causes a shift of the neighbouring structures. Thus, shift of the heart and the mediastinal organs, elevation of the diaphragm and shift and overinflation of the contralateral lung occur. If atelectasis of only one lobe is created, overinflation and eventually emphysema of the other lobes of the isolateral lung occur.

5. Experimentally all degrees of bronchiectasis, from slight cylindrical to most severe saccular forms, were produced when infections occurred in the bronchial tree of a lung whose bronchus was completely or almost completely occluded.

6. An infection that produced a purulent inflammation of the bronchi and only a desquamative-pneumonic exudate in the alveoli, was most likely to cause severe bronchiectasis.

7. The severest bronchiectases occurred within a period of about four weeks after the onset of the infection. If the survival period was several months, the bronchiectases were of more moderate degree.

8. Elimination of increase in the negative intrapleural pressure on the side of the atelectasis, by maintaining an artificial pneumothorax, had no effect in preventing or even moderating the development of bronchiectases.

9. Shrinkage of the lung following ligation of the pulmonary artery is not the same as pulmonary atelectasis. It is fibrotic organization of hemorrhagic necroses.

10. The roentgenologic signs of fibrotic shrinkage of the lung and atelectasis are the same.

11. Following ligation of the pulmonary artery no bronchiectasis developed, unless an infection intervened.

12. Following partial obstruction of the bronchus of a lung, patchy atelectasis, overinflation and emphysma of the lung occurred but no bronchiectases.

13. It is concluded from these experiments that the causation of bronchiectasis is inflammatory infiltration of bronchial walls with accumulation of exudate in the bronchial lumina; the latter is prevented from draining because of the artificially produced bronchial obstruction.

14. The possible application of these findings to human pathology are discussed briefly.

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DISCUSSION

DR. MAX PINNER, New York, N. Y.—I should like to take just a few minutes to reëmphasize a few points that seem to me to be of particular importance in the experiments reported, and to indicate what may be used in interpreting the pathogenesis of bronchiectasis in man.

First, atelectasis is not a necessary factor in production of bronchiectasis. Atelectasis is dependent on complete bronchial obstruction, while bronchiectasis is dependent on incomplete bronchial obstruction.

Second, the physical forces which are set free when a lung becomes atelectatic do not tend to dilate the bronchi. On the contrary, they tend to contract the bronchi for exactly the same reasons that they tend to contract the alveoli. This has been proved by the parallel series of animals which were kept under pneumothorax treatment.

The third point is that bronchiectasis in its pathogenesis depends on two separate factors; first, infection must be present which will weaken the bronchial wall by destroying all (in part, at least) elements, particularly the muscular and elastic coat. Secondly, there must be impairment of bronchial drainage, first in order to retain the infection to the extent that it may lead to destructive processes, and secondly, the stagnation of the exudate itself is probably one of the very important factors in dilating such bronchi.

The fourth point: The present roentgenological criteria for the diagnosis of pulmonary atelectasis are probably not correct, inasmuch as they do not differentiate between atelectasis and the secondary changes which may or may not follow atelectasis, such as fibrosis and bronchiectasis, suppuration and shrunken, fibrotic lungs.

Lastly it should be emphasized that there are, of course, important differences between animal experiments and human pathology. First of all, in these animals a normal bronchus was ligated suddenly. When a bronchus in the human being becomes obstructed, it is never a normal bronchus or a bronchus of a normal lobe.

Secondly, obstruction in man does not occur suddenly, and for this reason it is probably extremely rare in human pathology to see actually the development of pure, simple, uncomplicated atelectasis, and I have never seen it at autopsy.

The so-called postoperative atelectasis is most likely not a simple atelectasis, because there is no reason why atelectasis should make fever and leukocytosis as massive postoperative collapse regularly does.

Another point is probably that the physical characteristics of the chest wall in human beings, particularly in adults, are different from those in the rabbits; here the chest wall yields easily, so that the mechanical forces which are set free when a lung becomes atelectatic, are very much more easily compensated by the chest wall than is the case in adult human beings.

A METHOD OF TREATMENT OF LARGE PULMONARY AIR CYSTS (BALLOON CYSTS) BY AN ENDOCUTANEOUS FLAP

A. LINCOLN BROWN, M.D., AND WILLIAM BROCK, M.D. (BY INVITATION)
SAN FRANCISCO, CALIF.

THE vast majority of pulmonary cysts either pass unrecognized, or if diagnosed require no therapy. A certain number of cases demand intervention because they become infected, or because through a valve-like bronchial mechanism they become so distended they seriously decrease the patient's vital capacity. The methods of handling such conditions in the past have not been entirely satisfactory. The procedure described here was therefore evolved and in this instance proved to be efficient. Its rationale appears to warrant further use in similar cases.

Pulmonary cysts may be single or multiple, unilateral or bilateral. They may vary from a small or large isolated cyst to diffuse cystic disease, or so-called "honeycomb" lung. They may contain fluid or air; the latter may be expansile or nonexpansile. It is the large expansile isolated air cyst to which we have special reference. A good working classification of cystic disease is given by Sellors.[1]

"1. Solitary cysts—almost invariably large in size.
  "a. Huge 'balloon' or distension cysts in infants causing gross pressure signs and often death.
  "b. Smaller cysts about the size of an orange. These often remain clinically silent unless infected.

"2. Multiple cysts of varying sizes.
  "a. Medium-sized, in groups of two or three, close to the lung root on occasions. Size from a cherry to a golf ball. Usually clinically silent.
  "b. Small cysts, the size of a currant to a cherry.
    "1. Following a set distribution usually lobar in character. Conformity in the size of the cysts. 'Honey-comb' lung.
    "2. Diffuse or scattered lesions with cysts inconstant in size."

ANATOMY

Koontz[2] in 1925 recognized two general types of pulmonary cysts: (1) the bronchial dilatations with bronchial structures lining their walls, and (2) cavities resembling emphysematous blebs lying subpleurally.

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Read before the Twenty-fourth Annual Meeting of the American Association for Thoracic Surgery, Toronto, Canada, June 9, 10, and 11, 1941.

The greatest percentage of reported cases possess a lining wall of epithelium often ciliated, with underlying remnants of elastic tissue, smooth muscle, and cartilage. Others reported have a lining of flattened cells, or merely a hyaline wall.[3, 4] The histology may be greatly altered by the presence of infection. Occasionally viscid albuminous material containing epithelial cells and at times goblet cells may be aspirated from noninfected fluid cysts. The anatomy of the various types of cysts has been well described by Koontz[2] and more recently by Sellors.[1]

Many hypotheses as to the embryology and mechanism of formation of cysts have been suggested. These have been well reviewed.[1, 2, 5, 6, 29] Little has been added since the review reported by Koontz in 1925. Although many of these cysts may be no doubt acquired, the congenital nature of the majority is generally accepted. Schenck[7] reports thirty-six cases found at birth or in stillborn infants in a series of 368 collected cases, and a still larger number are found in infancy and early childhood. The type of epithelium and underlying structure in many suggests a congenital bronchial origin. Carbon pigment in the surrounding lung parenchyma is often conspicuously absent, suggesting lack of aeration of that section of the lung since birth. Associated pulmonary anomalies are a frequent accompaniment of cysts, and cyst cavities have been observed in anomalous pulmonary lobes.

Although the majority of these lesions are probably congenitally predetermined, their subsequent course depends primarily on the complicating factor of infection, or the presence and type of bronchial communication. Infection may produce clinical pictures easily confused with other types of pulmonary suppuration.

We believe that, in a sense, pulmonary cysts are essentially twofold in nature—(1) they are either primarily intrapulmonary, being located in the lung parenchyma or (2) they are found extraparenchymal, compressing the adjacent lung tissue. These latter tend to produce the balloon cysts and arise most likely from rests or herniations of the bronchi or trachea near the root of the lung. The first type might be best treated by lobectomy whereas the latter would appear to lend themselves well to extirpation. The clinical appearance and course of all cysts is naturally further determined by (1) presence or absence of infection within the cyst and (2) the presence or absence and type of connection with the tracheobronchial tree, be it open, closed, large or small, valvular or not.

The large expanding "balloon" cysts, as in the case reported, are without doubt associated with a check-valve or one-way bronchial communication, which may be intermittently open or closed. This type of valve mechanism has been well recognized in other pulmonary conditions[8] and has been described post mortem in cysts.[9, 10] These cysts are often clinically silent until this type of valve mechanism supervenes. Fluid cysts

have been known to empty their contents spontaneously, refill with air, and become expanding "balloon" cysts.[6] Certain observers state that air cysts contain fluid at birth, and their subsequent course depends upon the type of bronchial communication into the cyst.[5, 6] Anspach and Wolman have devised a classification of cysts depending on their postnatal behavior. Nonexpanding air cysts may be associated with a wide open bronchial communication that allows free ingress and egress of air, or so-called by-pass valve. Fluid cysts may remain filled only so long as a complete bronchial obstruction or stop valve mechanism exists.

DIAGNOSIS

The recognition of this condition is made difficult by its rarity. A clinical investigation of a case is usually begun because of the altered symptomatology brought about by infection or pressure mechanisms. The more common symptoms in a large series of collected cases have been enumerated by Schenck.[7] Cough, dyspnea, cyanosis, fever, and hemoptysis are most frequently observed. These symptoms are brought about by either infection or lowered vital capacity associated with increased intracystic pressure, or both. The large expanding air cysts produce a clinical picture closely resembling that of a pressure pneumothorax, with recurrent progressive attacks of severe dyspnea. The great majority of the reported cases have been observed in infancy.

The diagnosis rests mainly on the roentgenologic appearance of the lesion. Obviously because of the many types of cysts, single, multiple, fluid or air containing, and always possibly altered by the presence of infection, the roentgenologic appearance may be varied. The roentgenologic differential diagnosis has been well presented.[11] An air cyst may vary from a small area of decreased density or a curvilinear area of increased density, denoting the cyst wall, to a large air space occupying one whole side of the thorax and encroaching on the other. Differentiation from a pressure pneumothorax is often difficult as was true in the case herewith reported. In pneumothorax it must be remembered that the air is in the pleural space. It exerts uniform pressure and collapses the lung toward the hilum. The intracystic pressure of the balloon cyst is within the lung and exerts pressure on the lung in all directions. Hence areas of atelectatic lung may produce areas of density peripherally, at the apex, cardiohepatic angle, and the costophrenic sulcus. Typically in pneumothorax the lung shadow is compressed to the hilum while the other above-mentioned areas are clear. In addition fine trabeculations traversing the cyst cavity may be frequently made out. Occasionally the cyst wall may be made out, especially if there is an accompanying pneumothorax.

Various special methods of study are available, although the usual dangers are present, especially in the case of air cysts. The use of

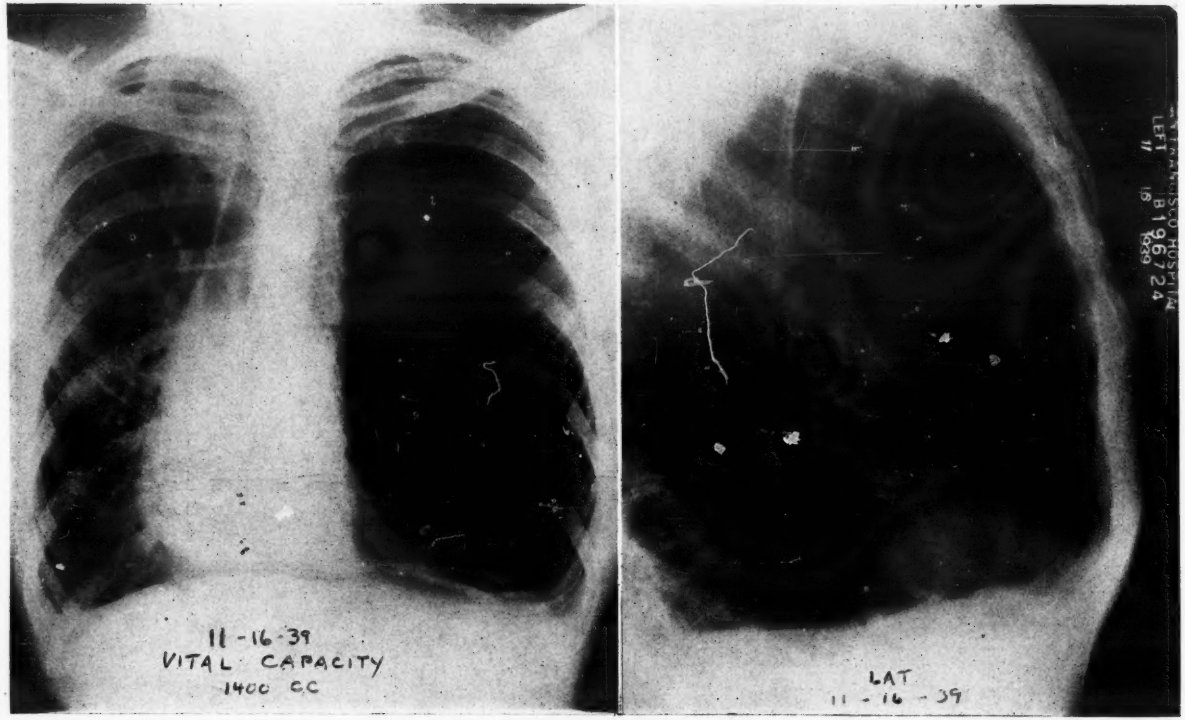

Fig. 1.—Nov. 16, 1939. Roentgenologic appearance of chest on admission of patient, showing entire left chest to be filled with air. The trachea, mediastinum and heart are displaced to the right with a complete collapse of the left lung.

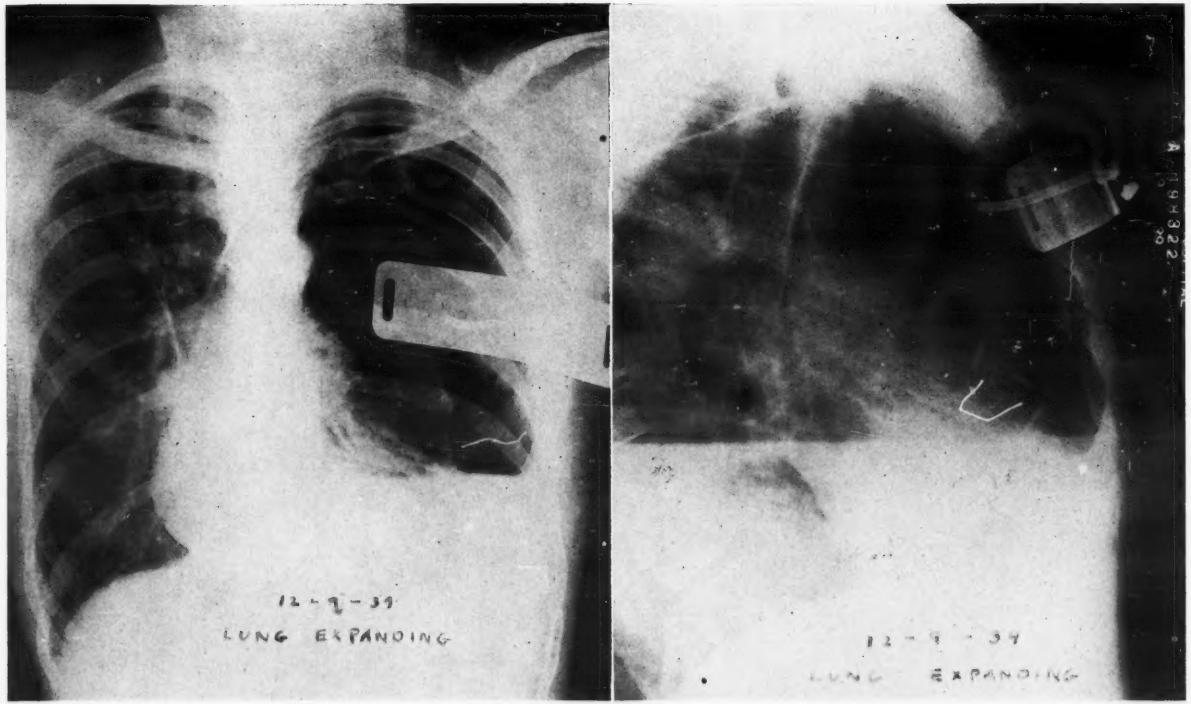

Fig. 2.—Dec. 9, 1939. The metal cannula has been introduced. The mediastinal shift has decreased; the left lung is expanding; and there is some fluid in the air space. (This is the space which was subsequently shown to be inside the cyst.)

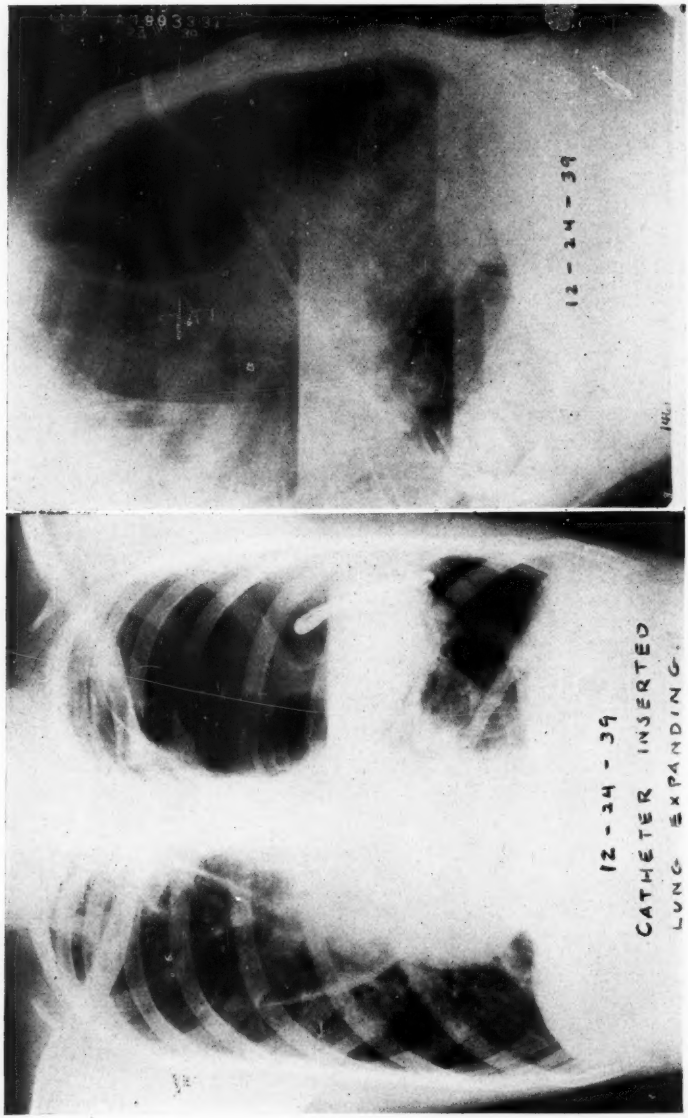

Fig. 3.—Dec. 24, 1939. There is further expansion of the lung and the metal cannula has been replaced by a rubber catheter.

iodized oil injected into the tracheobronchial tree or directly into the cystic cavity in the larger cysts has been of definite diagnostic value. Intratracheal lipiodol will enter the cyst cavity only if the bronchial communication is patent. Various roentgenologic views with the patient in different positions may show fluid levels and outline the cyst wall. From the intracystic pressure readings one may make certain assumptions about the patency and type of bronchial communication. Thoracoscopy may visualize the cyst wall and occasionally its bronchial opening. Biopsy of the lining membrane may yield a histologic picture previously described. From fluid cysts the typical viscid, cell containing fluid may be aspirated.

THERAPY

The treatment of pulmonary cysts depends on the type of the disease and the stage in which the condition presents itself. To eradicate the disease completely the entire secreting cyst wall must be removed. Enucleation is generally nc feasible because characteristically the surrounding lung parenchyma blends with the cyst wall and no cleavage plane can be obtained. Single or multiple cysts isolated to one lobe have been successfully removed by lobectomy on several occasions, but this usually presupposes the removal of potentially available lung tissue. For the diffuse honeycomb bilateral disease there is no adequate therapy. The usual conservative measures as employed for bronchiectasis have been employed.

The presence of infection has often made external drainage mandatory; however draining external fistulas result unless the cyst wall is removed.

Transthoracic closure of the bronchial opening in "balloon" cysts has been apparently successful in at least one instance and has been attempted in others.[12]

Collapse therapy as employed in tuberculous cavities has been used in a few cases with no apparent beneficial results.[13] This procedure is obviously not physiologic when a stop-valve or a check-valve bronchial communication is present.

Improvement has been noted after the intracystic instillation of lipiodol, probably resulting from inflammatory closure of the bronchial opening and a cessation in the pressure mechanism.[12, 14, 15]

The "balloon" or expanding air cysts offer a special field for therapy. Primarily their symptoms result from their peculiar type of bronchial valve mechanism. Complete removal of these cysts is generally not surgically feasible, except by pneumonectomy or lobectomy. The check-valve communication and the resulting symptomatology could be relieved mechanically by closure of the bronchial opening. This has been attempted surgically,[12] and the use of sclerosing solutions has been

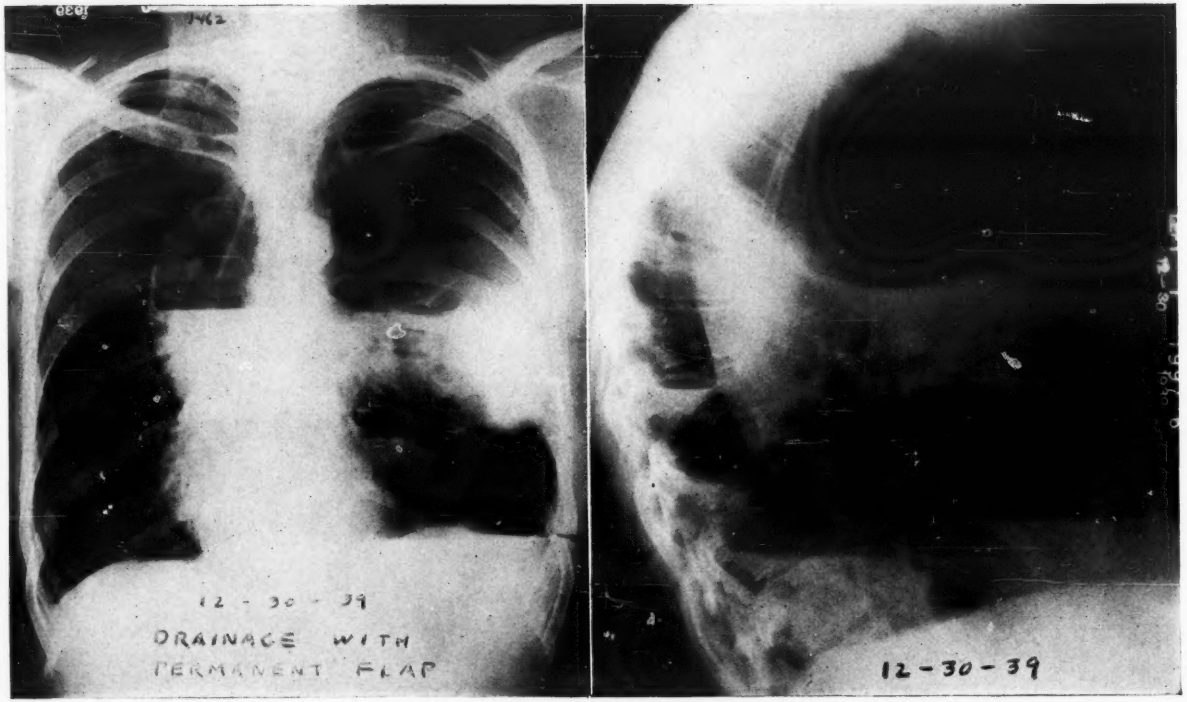

Fig. 4.—Dec. 30, 1939. The endocutaneous flap has been made; expansion progresses. There is now a herniation from the left to the right; this persisted for only a short time.

suggested by Adams in a personal communication to Pearson.[13] There are many reports in the literature of only temporary relief of symptoms following thoracentesis and the release of positive pressure in infants.[1, 6, 7, 13-15] Temporary tube drainage with the use of a flapper valve has been attempted with alleviation of symptoms.[3]

DISCUSSION

In the case reported we were dealing with a "balloon" type of cyst with a valve action in which the patient became progressively more dyspneic with each attack. One of these attacks was temporarily relieved by needle aspiration of the excess air from within the cyst. Therefore it occurred to us that, if a permanent external valvular exit were made for the egress of air to the outside, the acute symptoms due to the distension of the cyst might not only be relieved but would be prevented. Furthermore, by such an external pressure release the cyst might collapse and the potentially available surrounding lung might expand and become functionally available. Such was found to be the case.

Because of the collapse of tuberculous tension cavities with suction drainage, temporary drainage with continuous suction was instituted in this case. This was accomplished by introducing a cannula directly into the cyst cavity. This was followed by a gradual collapse of the cyst and a concomitant expansion of the surrounding lung tissue. The beneficial results noted from this procedure pointed to the advisability of maintaining such permanent air drainage. Therefore an endocutaneous flap was made directly into the cyst, connecting the cyst to the outside. This proved satisfactory in that the positive pressure within the cyst was eliminated and the surrounding lung tissue expanded and became aerated. The flap was made according to the method suggested by Eloesser[16] in the drainage of certain empyema cavities.

CASE HISTORY

J. E., a 41-year-old draftsman, entered the San Francisco Hospital on Nov. 15, 1939, complaining of progressive severe dyspnea for a period of two weeks. He gave a history of fourteen years of periodic attacks similar to the present, which were characterized by gradual increase in shortness of breath over a period of five days to two weeks. Each attack would culminate in an episode of extreme dyspnea which would completely incapacitate him for a period of two to four days. During the first years after the onset the attacks would come on at intervals of every six months to a year. They gradually increased in number and severity until the eight months before entry during which time he had six attacks. The dyspnea was frequently associated with a swelling of his hands and feet and marked cyanosis. He had never noticed wheezing, hemoptysis, coughing, or chest pain. Between attacks he was moderately incapacitated by dyspnea but managed to carry on his usual activities. Two weeks before entry he began to become increasingly dyspneic, particularly at night when he would awaken gasping for

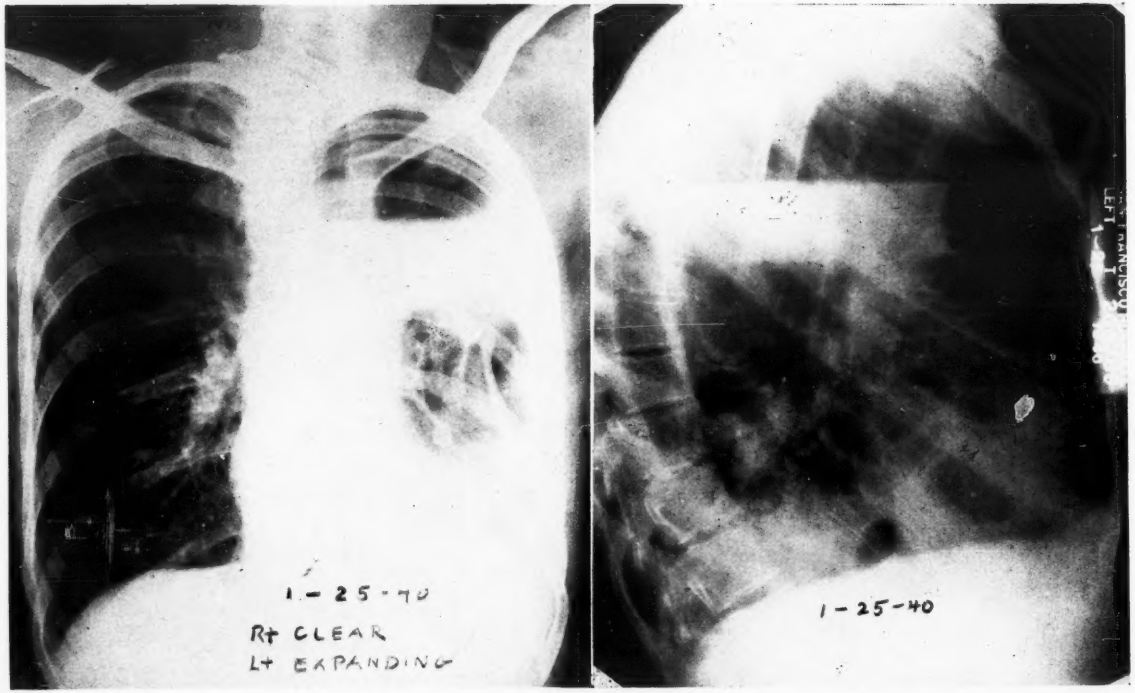

Fig. 5.—Jan. 25, 1940. The further expansion of the lung and the diminution of the cyst cavity show the endocutaneous flap to be functioning well.

breath. The day before entry he became markedly dyspneic and collapsed. He was found almost "black" with cyanosis.

Since the age of 11 he had suffered from bronchial asthma periodically, especially in the fall and in the past had been found sensitive to many pollens. During the last ten years this had completely disappeared. At five years of age he had pertussis and had pneumonia at 27 with no known complications. His father died of bone tuberculosis before any contact with the patient.

Fig. 6.—Feb. 28, 1940. This photograph shows the present drainage opening produced by the endocutaneous flap.

Physical Examination.—Revealed a severely dyspneic male, moderately cyanotic, with movements of the accessory muscles of respiration. The thorax was of a markedly emphysematous type with little respiratory excursion and less on the left side. Percussion note was resonant throughout. Breath sounds were absent on the left and present but diminished on the right. There were a few tubal râles scattered through the right lung field. Examination of the heart found the apex beat in the right anterior axillary line in the fifth intercostal interspace. The cardiac sounds were of good quality, of regular rhythm, and no murmurs were heard. No sounds were detectable at the usual aortic and pulmonic areas. The trachea was markedly deviated to the right. There was no clubbing of the fingers. The remainder of the examination was essentially negative.

Laboratory Date.—The electrocardiogram showed slight right axis deviation. The urine was negative except for eight or ten pus cells per HDF. Blood analysis: Hemoglobin was 108 per cent, 15.7 Gm.; R.B.C., 5,000,000 per cubic centimeter; W.B.C., 11,400; filament count, 72 per cent; nonfilament count, 8 per cent; lymphocytes, 16 per cent; monocytes, 4 per cent. Wassermann and Kahn reactions were negative. The sputum was negative for tubercle bacilli.

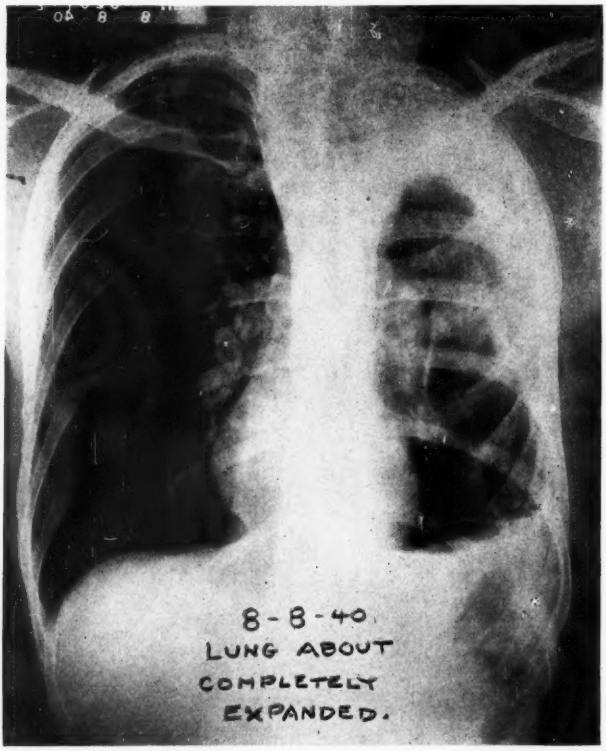

Fig. 7.—Aug. 8, 1940. Almost complete expansion of the left lung with marked pleural thickening of the left apex. At this time there was essentially no drainage. The patient gained about 20 pounds. Vital capacity was 2,200 c.c.

On the night of entry to the hospital the patient became increasingly cyanotic and dyspneic to the point of becoming irrational. A needle was inserted in the left second interspace anteriorly, and a considerable amount of air under positive pressure was released. No pressure readings were obtained. Following this procedure the patient was relieved and went to sleep. Five days later pressure readings were taken. The original readings were minus 2 and plus 2. Following the insertion of 200 c.c. of air the readings remained the same. After the removal of 300 c.c. of air the readings were still the same.

On December 1 a thoracoscopy was done. When the thoracoscope was inserted, air gushed out under positive pressure; however toward the end of the procedure the pressure became neutral. The lining membrane of the cavity appeared white and smooth without any unusual contour, appearing very much like the surface of

an ovarian cyst. It was difficult to determine whether the surface visualized was the pleura or lining membrane of a cyst. A trocar was left in place and a constant negative pressure maintained until December 18, when, because of infection in the cyst space and discomfort caused by the cannula, it was replaced by a rubber catheter.

A moderate amount of brownish sanguineous, seropurulent drainage continued to come from the catheter which yielded a mixed infection on culture, and on two occasions sterile serosanguineous fluid was obtained low down from the pleural space proper by thoracentesis. The patient's fever continued to reach 102° to 103° F. rectally daily.

On December 27, an endocutaneous flap operation into the cyst cavity was performed. This was followed on subsequent days by excessive seropurulent drainage from the cyst cavity. The patient's fever again reached as high as 103° F. rectally, for the following ten days but gradually subsided to normal coincidently with sulfanilamide administration and blood transfusions. A biopsy of the cyst wall failed to reveal any epithelial lining, cartilage, or elastic tissue and showed only chronic inflammation.

The patient was discharged on Feb. 20, 1940 remarkably improved, ambulatory, and having no respiratory discomfort. The vital capacity had measured from 1,400 to 2,000 c.c. Three months later he had gained twenty pounds; the flap was working well and discharging only about 1 ounce of sanguineous material daily. He has had no subsequent attacks of dyspnea and is able to carry out his ordinary work without discomfort. He states he has much less respiratory difficulty than had been present in the previous ten years.

The Figs. 1-7 show the gradual change in the roentgenologic appearance of the lesion.

DISCUSSION

Massive "balloon" cysts are a peculiar surgical problem. The removal of the cyst itself is often a hazardous procedure. The cyst, if intraparenchymal, may be removed by lobectomy or pneumonectomy, but this presupposes the removal of potentially functional lung tissue. The collapse of the cyst by an external valve as described here not only alleviated the patient's symptoms but preserved for this patient potentially active tissue. The importance of infection and pressure mechanisms must be appreciated.

SUMMARY

The anatomy and physiology of "balloon" cysts are briefly reviewed.

A method of treatment of massive "balloon" cysts by the use of an endocutaneous valve flap is presented. A case in which it was employed successfully is reported.

The use of the endocutaneous valve flap is suggested in other cases of "balloon" cysts when radical excision of the cyst wall may not be deemed the method of expediency.

ADDENDUM

The patient remains well with essentially full expansion of the lung. He is doing heavy manual labor, working as a truck driver.

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DISCUSSION

DR. CAMERON HAIGHT, Ann Arbor, Mich.—The subject of pulmonary cysts is a fascinating one, especially as the individual cases differ so greatly in their various aspects. One of the important points that must be determined in the management of cysts is to ascertain whether they have or do not have an epithelial wall.

Dr. Brown has presented an ingenious method for the treatment of large balloon cysts. Before discussing the treatment of balloon cysts, I would like to refer to six patients with epithelial cysts of the lower lobe upon whom Dr. Alexander and I have performed lobectomies. Two of these cases have been reported by Dr. Maier and myself. Clinically, they resembled large empyemas. The remaining four cases did not simulate empyemas. Two of them closely resembled large pulmonary abscesses, and in one of these cases the diagnosis was not definitely established until the pathologic examination of the cyst wall was obtained, revealing its epithelial lining. It is perhaps a coincidence, but it is of interest that two of these six patients with lower lobe cysts had a large anomalous pulmonary artery

[as described by McCotter (1910) and Batts (1939)] coming through the diaphragm and supplying the lobe containing the cysts. The anomalous artery in each of the two cases was larger than the diameter of an ordinary lead pencil. Whether these epithelial cysts, which were probably congenital in origin, and the congenital arterial anomaly are directly related, is an interesting speculation.

I should like to refer now to a case of a very large acquired balloon cyst treated by pneumonectomy on Sept. 8, 1938. A review of the literature reveals only one similar case of tremendous balloon cyst occupying the entire hemithorax and treated by this method, a patient operated upon about four years ago by Mr. J. E. H. Roberts. We are not certain that a pneumonectomy is the procedure of choice in balloon cysts. In the case to be described, the pathologic report revealed the presence of a nonepithelial wall. Had we been aware of this fact in advance of operation, the possibility of tube drainage and subsequent suction would have been given more serious consideration. The original plan in this case was to remove the cyst by means of a lobectomy, but for technical reasons, it was necessary to resort to pneumonectomy.

(Slide) The first roentgenogram (Fig. 1*A*) of this patient was made during an illness from scarlet fever and pneumonia in another hospital. A basal empyema is present, and there is beginning rarefaction in the axillary segment of the left lung.

(Slide) The next roentgenogram (Fig. 1*B*), made one month later and following drainage of the empyema, demonstrates an increase in the size of the rarefied area in the axillary segment of the left lung.

(Slide) Three and one-half months later (Fig. 2*A*) one sees a large acquired cyst with superimposed ridges of pulmonary tissue. This roentgenogram is rather typical of a large cyst because of the presence of these ridges. The empyema at this time was healed.

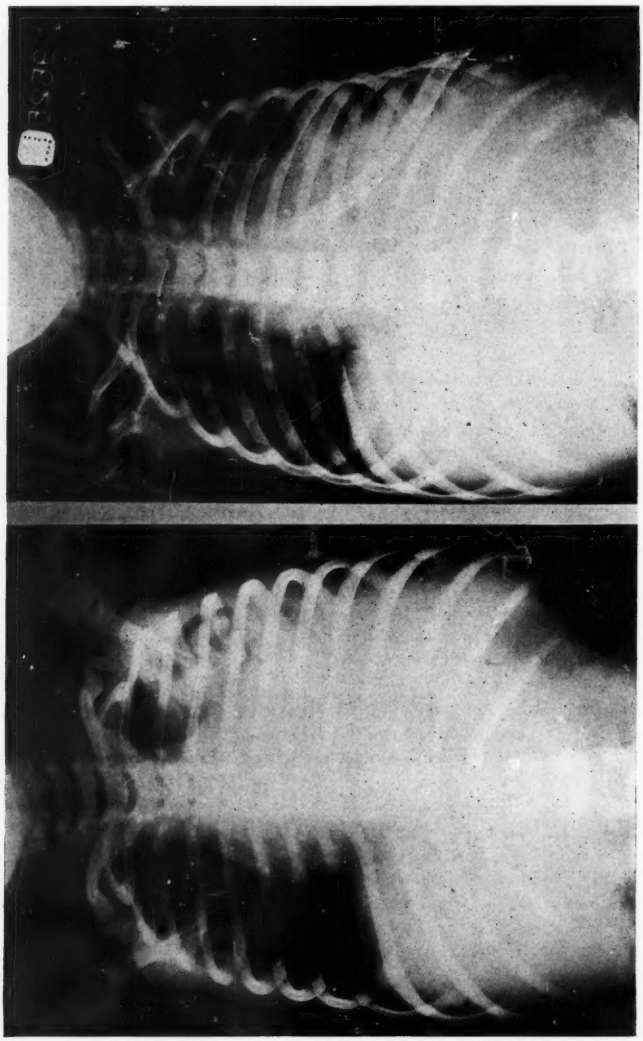
(Slide) A roentgenogram (Fig. 2*B*) made approximately one year later, at which time we first saw the patient, demonstrates further enlargement of the cyst. As the empyema had undoubtedly caused a fusion of the pleurae, a determination of the intracystic pressure was considered safe in this case. This was done, and the positive pressure within the cyst was so high that the water was expelled from the manometer.

We next investigated the interior of the cyst with a thoracoscope, as did Dr. Brown. It is interesting to note that the portion of cyst wall covering the aorta was so extremely thin that it could not be seen, the aorta appearing normal, as if no cyst wall covered it.

It was hoped that during the thoracoscopy the bronchial fistulae could be visualized and cauterized. In the event that this was not feasible, we at least hoped, in view of the acquired nature of the cyst and the probable absence of epithelium in its wall, that the thoracoscopy would precipitate an effusion with a deposition of fibrin that would seal over the fistulous openings. As the fistulae were buried in deep recesses, it was not possible to visualize them, and an effusion did not occur within the cyst following the thoracoscopy. A biopsy of the cyst wall was contemplated, but the thinness of the cyst wall did not allow one to be obtained with safety by the thoracoscopic method.

(Slide) A subsequent roentgenogram shows further enlargement of the cyst, although the patient's only symptom at this time was mild dyspnea. It was then decided that the cyst should, if possible, be removed by lobectomy. At operation, however, an adequate cleavage plane did not exist between the upper lobe from which the cyst arose and very contracted atelectatic lower lobe, and so, pneumonectomy seemed the safer operation. Also, at the time, it seemed doubtful that

the contracted lower lobe could have been expanded to fill the pleural space, had a lobectomy been possible. As there was no reaction around the hilar vessels, individual ligation of the pulmonary vessels and primary suture of the bronchus could be readily carried out. The patient's course subsequent to the pneumonectomy was uneventful and the wound healed per primam.



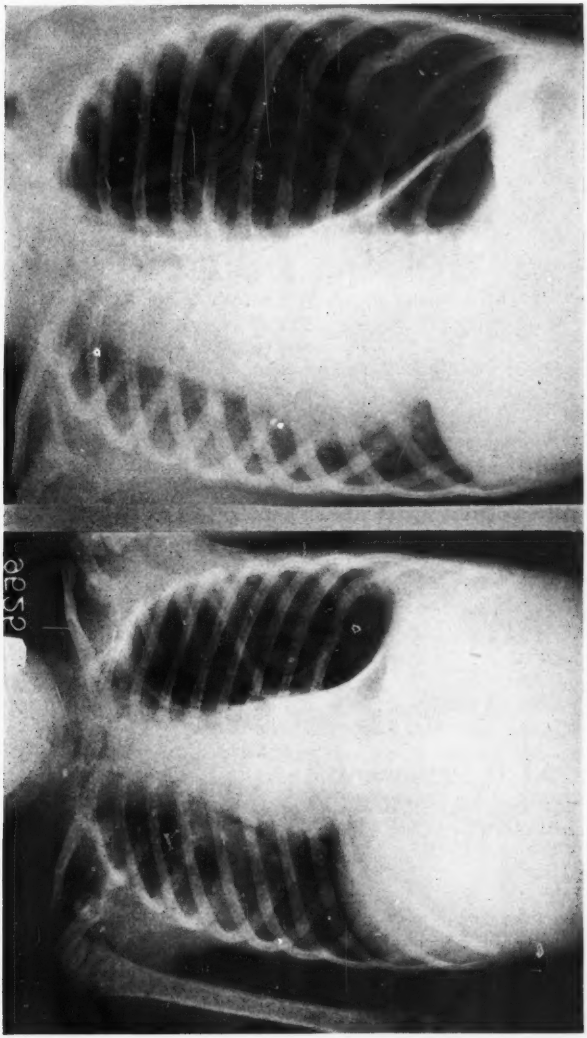
B.

A.

Fig. 1.—*A*, April 28, 1937; *B*, May 29, 1937.

(Slide) The next roentgenogram shows the postoperative condition of the chest following pneumonectomy. No empyema developed. The mediastinal structures are now retracted to the left and the pleural space is obliterated by serum and fibrin.

(Slide) A photomicrograph of the cyst wall reveals columnar epithelium only at the site of the fistula. The other photomicrograph demonstrates the nonepithelial character of the cyst wall proper.



B.

A.

Fig. 2.—*A*, Sept. 8, 1937; *B*, Aug. 20, 1938.

The method which Dr. Brown has presented for treatment of large pulmonary air cysts by an endocutaneous flap possesses the advantage of conservation of pulmonary function. The operation of lobectomy or pneumonectomy appears to be a more certain method for eradication of an epithelial walled cyst, although it involves the sacrifice of pulmonary tissue. If the case which I have presented were to be treated at the present time, we would undoubtedly use the method of drainage in preference to pneumonectomy, in view of the nonepithelial nature of the cyst

wall. However, I feel that we have not yet had sufficient experience with this type of cyst to know exactly how it should be treated. Dr. Brown's contribution is important in adding a new measure to the surgical armamentarium.

DR. W. E. BURNETT, Philadelphia, Pa.—Since there is so little written about this subject, it seems worthwhile to discuss three cases in which we have done one method of excision, and one, which I would like to demonstrate, in which we used another.

I enjoyed Dr. Brown's instructive paper very much, and the last case I want to show is similar to the last one that he demonstrated.

(Slide) Our first three cases were infected. We drained them primarily, and in a period of from three weeks to two years we went back and excised the cyst wall. All these patients made nice recoveries following excision, and there was quite rapid healing. This last case is somewhat similar to the subject under discussion.

(Slide) This patient was a man of about 45, who had been quite a smoker. He had had symptoms for a period of a year. These consisted mainly of slight cough, and some dyspnea.

Dr. Gary, who is here in the audience, also saw this patient and made several pressure readings. It was found that the pressure was neutral to slightly positive. After aspiration there would be some re-expansion of lung as indicated in this first film, but in a few hours the pressure would return to the original state. At no time was there marked positive pressure producing a shift to the other side.

After considerable effort to aspirate this or to find the bronchial openings, it was decided to do a lobectomy on this patient.

(Slide) However, on entering the thorax, we found an anomaly which I gather was suggested in Dr. Haight's discussion this morning. In this upper lobe lesion, there was no anomalous vessel, but a fusion of the middle and upper lobes. There was absolutely no division between them and all that portion of the lung was atelectatic. There was aeration of a small portion of the lower lobe.

It was feasible to excise this cyst. It had shown no infection previously, so we went ahead with the primary excision. We followed the plane of cleavage without much difficulty and very little bleeding. The result was this specimen which you see here, with marked trabeculation inside, visible by thoracoscopy. The outcome was very satisfactory.

This film was made a few days after operation, and the other one at a period of about three and one-half months. Unfortunately, we have no recent one, but the patient has continued well for about a year.

Thus it seems possible, in the absence of infection, to excise such cysts, retaining all of the normal part of the lobe or lung, and thus re-establish function by re-expansion of the conserved normal portions. With proper control of the blood supply by a loose tourniquet about the hilus which can be tightened if dangerous bleeding occurs, there seems to be only moderate risk. Of course, if the cyst is infected, primary excision should be a very dangerous procedure, but a two-stage operation with drainage as the first stage followed at an appropriate time by secondary excision seems to eliminate most of this risk.

This has seemed the preferable method of handling such cases, but I am very glad to hear about other methods which may come in handy at other times.

DR. DEAN B. COLE, Richmond, Va.—We have had one patient, a teen-aged girl, operated on by Dr. Johns for large pulmonary air cysts, which had become infected. There has been no further cystic development so we feel the operation was indicated and successful. We have also had five patients die as the result of cystic lung disease, and autopsy in each case revealed multiple cysts. We are convinced that operation was not indicated in any of these five patients. Until we

have more autopsy studies of cystic lung disease, any selection of cases for surgery must necessarily be a haphazard procedure. Since this condition becomes manifest so often in childhood, it should be borne in mind by both the pediatrician and roentgenologist.

DR. EDGAR W. DAVIS, Washington, D. C.—The more I study pulmonary cystic disease, the more I feel as I did yesterday about the various discussions on closures of the bronchi in lobectomies and pneumonectomies—the more I study, the less I know.

About six or seven years ago, I think, Dr. Joseph Gale did a pneumonectomy for cystic disease, and that was the first cystic lung I knew of that produced sufficient symptoms to justify pneumonectomy. About a year or so after that, Dr. Alexander removed a lung in a 14-year-old boy for cystic disease.

In 1936, just before leaving Ann Arbor, we had a case we thought was an empyema, which I drained, and which turned out to be an infected cyst. I understand Dr. Haight subsequently did a lobectomy.

I was very much in hope that someone in the discussion of this subject would give us some idea as to what role an obstructed bronchus may possibly play in producing a cyst, and describe the mechanism of its formation, and tell us whether or not these are congenital cysts or acquired cysts.

Some of them, it is true, are epithelized; others are not. The majority have been infected—at least, they are when I see them. But in spite of the fact that they are tremendously infected, the histologic sections of the walls of these cysts do not represent true inflammatory tissue. In fact, those that are not epithelial cysts do not show true inflammatory tissues in the walls.

I did a lobectomy on a 37-year-old man who had been a star football player some eighteen or twenty years ago. His only symptom was cough and shortness of breath. He had a perfectly huge balloon cyst 12 or 15 cm. in diameter, which was surrounded by probably twenty-five to fifty very small cysts.

A lipiodol bronchogram did not visualize oil within the cyst, that is, the bronchi were completely obstructed. Removal of the lobe and subsequent suture of the bronchus going into the lobe was done. A small needle was used, and an attempt was made to force fluid into this cyst. No fluid went into the cyst. In other words, there was a complete obstruction. We could not insert an aspirating needle with a 50 c.c. syringe and force air from within the cyst outward through the bronchi.

It would seem that there was an obstruction to the ingress and the egress of air from that cyst, yet air had gone into the cyst. How it got there, I cannot say. We subsequently attempted to force some very thin barium paste into the cyst, and we took roentgenograms at different degrees of pressure. The barium did not enter the cyst; it merely diffused around the epithelized wall of the cyst.

We still do not know whether that was a congenital or acquired cyst, but it is presumably congenital.

One of the best cases we have had was a huge cyst of the left upper lobe in a girl 19 years of age, who had been coughing at intervals for some eleven or twelve years. She had an extensive bronchiectasis in the left lower lobe.

We debated as to whether we should drain that as an abscess or a cyst, and make a subsequent removal of the lower lobe for bronchiectasis. We took pressure readings and we found a −2 to a +2. After opening the chest, we found an inflammatory obliteration of the interlobar fissure which prohibited lobectomy.

Inasmuch as she had disease in the lower lobe, we proceeded to do a pneumonectomy instead of a lobectomy. She has made an uneventful recovery. Upon dissection of the bronchi, again we could find no evidence of a freely communicating

bronchus with the cyst, but we were able to find a small piece of chicken bone obstructing the bronchus that presumably has been there for some eleven or twelve years.

We had another patient for whom we did an upper lobe lobectomy for profuse hemorrhage. In fact, his hemoglobin was only 20 per cent. We gave him 4,500 c.c. of blood, and through a paravertebral nerve block, opened his chest and got a tourniquet around the hilum of his upper lobe; we then gave him intratracheal anesthesia and successfully removed the lobe.

Again, this patient's bronchus was almost completely obstructed by a benign papilloma in the bronchus draining the cyst.

We had another patient who had had an infected cyst for some thirty-three years. She was 42, I believe, at the time of the operation. Again we could find no communicating bronchus. We did a lobectomy on her and had a tremendous amount of difficulty in removing the cyst from the diaphragm.

Since Dr. Haight has called attention to this anomalous vessel coming up through the diaphragm, I feel reasonably sure that we encountered in this case that same anomaly, although at the time I did not recognize it. We had considerable bleeding, but it was effectively controlled. Due to a surgical blunder, she died some six or seven days postoperatively of a pericarditis.

DR. RICHMOND DOUGLASS, Ithaca, N. Y.—Lobectomy for infected isolated cysts in the lower lobe has on three occasions revealed the presence of an anomalous vessel. In the first instance, a girl of 15, the vessel came up from the lowermost angle and entered the lower portion of the left lower lobe. Whether it pierced the diaphragm or came off the aorta at a low level, we were not able to determine. In the next instance, a young woman of 22, the vessel lay in adhesions which were in approximately the position of the lower border of the pulmonary ligament. In the third case, a 20-year-old woman, the vessel was in a similar position. It happens that in each instance the left lower lobe was involved. Whether this is of significance we do not know. The important thing is that, when an operation is undertaken for a pulmonary cyst, the possible presence of an anomalous vessel should be suspected and injury to this vessel avoided.

DR. A. LINCOLN BROWN.—I wish to thank the men for their discussions and their contributions and to reiterate that the procedure suggested here is not for all cysts. It may be of value in balloon cysts, particularly if they are large and infected.

SURGERY OF THE CHEST IN WAR

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TORONTO, CANADA

IT IS unnecessary before this society to review the epochal development of thoracic surgery during the last two years of World War I, much less the great strides that have been made in surgery of the chest since those fateful days. Suffice it to say that thoracic surgery has been made safe, and it solely remains to make patients safe for thoracic surgery—that in war means immediate recognition of the nature of the wound, prompt attention, early evacuation to a surgical center, resuscitation and measures to offset shock, control bleeding, prevent infection, and to make possible surgical interference at the earliest possible moment.

Experiences in Spain and now in the present war with attacks on civilians in villages and towns and large crowded centers of population in cities reveal somewhat different types of chest wounds from the majority seen during World War I. The great increase in machine-gun fire, the vastly greater intensity of the bursting effect of the modern bomb, the intense antiaircraft fire, the fire bomb, and the number wounded by falling masonry and flying fragments cause a higher percentage of immediately fatal chest wounds and a greater percentage of crushed and stove-in chests than were seen in the past.

Mercer (Edinburgh) states that of 1,000 casualties in the bombed area, 600 are dead, 300 are gravely wounded and require immediate attention, and 100 are lightly wounded and can walk away.

Types of wounds of the chest in air raids are (1) stove-in chests from falling masonry, (2) bomb wounds from shrapnel, fragments and flying missiles, (3) bullet wounds from machine guns of low-flying aircraft, (4) shrapnel from antiaircraft guns, and (5) collapse of the lung from perforation, laceration, or what-not from the force of the "blast."

The immediate mortality from the force of the "blast" particularly on hard surfaces, such as pavements, docks, and the decks of ships, is very high. In addition, the relative percentage of multiple wounds in the chest and elsewhere is higher than in ordinary fighting. The mortality from chest wounds is very high among women and children.

TYPES OF WOUNDS

The types of chest wounds are similar to those of the last war. They are (1) injuries to the chest wall, with or without damage of the thoracic

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contents, (2) perforating, "E and E," (3) penetrating "E only," (4) abdominothoracic, and (5) collapse of the lungs from "blast" without an actual wound of the chest wall or a serious wound elsewhere.

It must be constantly kept in mind that a missile may enter almost any part of the body and traverse or lodge in the chest.

Partial or Complete Collapse of the Lung Without a Wound.—Early in July of 1918 we recognized, for the first time, collapse of one or both lungs as a cause of death in dead and dying men without any apparent wound. This occurred among wounded in close proximity to a shell or bomb blast, and has been more common in the present type of warfare. A traumatic asphyxia occurs usually associated with rupture of the visceral surface of the lung and perhaps with minute petechial hemorrhage into the lung tissue itself. However, the final word has not been said on this type of death. The author has gone into this question at length in previous articles. A condition similar to the bends or caisson disease may account for a definite percentage of such deaths.

The parietes may be gravely damaged by falling masonry or a large fragment of shell casing without any serious, apparent external wound. Involvement of the bony wall of the chest may be complicated by spinal injury, either fracture-dislocations or crush fractures.

Internal injuries such as hemorrhage, a ruptured trachea, bronchus, esophagus, or collapsed lung must be sought.

Surgical emphysema is more common in these crushing and "blast" injuries of the chest. It is alarming to a conscious patient but in itself is not serious. It must not be confused with the crepitus of gas gangrene.

The value of roentgenologic examination in chest wounds will be dealt with later.

Many valuable contributions have been made by various authors on the variety of chest wounds observed in the last war, particulars of entrance and exit wounds, the extent of bony involvement, retention of missiles, damage to the parietes, the lungs, the pericardium and heart, the liver and diaphragm, the trachea, esophagus and aorta, the complication of multiple wounds, involving both chest and vital parts elsewhere, the significance and extent of hemothorax, and the role of shock, infections and contralateral collapse. Such should be the common knowledge of every physician and surgeon called on to deal with thoracic wounds. The bizarre group of physical findings in a closed and open chest wound and after injuries to the heart and pericardium should be recognized. These problems cannot be dealt with in the scope of this contribution.

Variations in penetrating wounds of the chest in the last war and in this war are being found. Hospital staffs in large urban centers are being faced with the type of wound seen only in advanced dressing

stations and casualty clearing stations in the last war, on the Western front. Cases are being seen earlier before hemorrhage, shock, exposure, and infection are established. Gas gangrene should be less prevalent than in the heavily manured fields of France and Belgium. Infection generally should be less common as patients are seen earlier. Skilled thoracic surgeons, special chest centers, and mobile units should be available. With the vicious type of explosive now in use there is more burning and searing of the tissue than in the last war and probably, as a result, less infection. This escharotic effect is to a certain extent offset by the great tearing and bursting effect of the fragments of the high tension bomb.

PROTECTIVE MEASURES

Antitetanic inoculation, antigas gangrene injection, and the use of sulfanilamide in wounds and by mouth, undoubtedly, should reduce the incidence of grave infections and complications.

Sulfanilamide powder should be of great value in chest wounds and should reduce the percentage of infected hemothoraces that contributed to so many deaths.

BLOOD TRANSFUSION

The great availability of blood for transfusion and simpler methods of storing, transmitting, and administering it should greatly reduce mortality.

X-RAYS

In this war it is to be hoped that adequate roentgenologic facilities will be available in all theatres of war. It might be well to point out that until late in the fall of 1916 we had no x-rays in use on the Somme, and yet our mortality after we began to operate upon the so-called fatal chest wounds was not much greater than in the later months when x-ray units were supplied. Therefore, if in the stress of fighting x-ray units are not available, the thoracic surgeon must not delay surgical interference for that group of patients who assuredly will die within the first forty-eight hours if not adequately dealt with surgically.

However, it is tremendously important and valuable and even life saving in many patients to know the exact position of a retained missile, fragments of bone prior to operation. The simple method of localization of missiles perfected by Donaldson, Finzi, and others in the last war should be adopted by all roentgenologists. Missiles can be localized rapidly with the greatest of accuracy and without disturbing patients to any great extent. The eminently practical type of mobile x-ray units now in use should be made available in all surgical centers.

Even now twenty-three years after the last war we are embarrassed by the lack of x-rays at that time, and we have recently seen a man with a bullet embedded in the heart since 1916. He has been an invalid ever since, but for over twenty years was looked on as a scrimshanker and allowed a pension of 12 per cent only. He has dyspnea on the least exertion, frequent so-called "fainting" spells, and cannot exert himself to any extent without bringing on such an attack. He has no precordial pain of any consequence and thus far has refused to permit removal of the bullet.

ANESTHESIA

Greater refinements in methods of inducing anesthesia should materially contribute to a lower operative mortality in chest wounds in this war. Intravenous anesthesia, such as pentothal, will be of value as will greater refinement in intratracheal anesthesia. Paravertebral and local infiltration still will have its place. Spinal anesthesia will be of value in dealing with abdominothoracic lesions.

In this connection I might point out that a year ago John Lundy (from the Mayo Clinic) said to me, "Lockwood, out of the last war came thoracic surgery, out of this war will come anesthesia," and I have a great regard for his judgment.

INCIDENCE OF CHEST WOUNDS AND MORTALITY

It is difficult indeed to predict the relative incidence of chest wounds that will occur and the mortality to be expected. From my experiences in five years of war service in World War I, and from statistics of the Spanish revolution and the past twenty months, I believe we shall have a higher percentage of chest wounds to deal with surgically than in the last war. More immediate deaths will occur and it is a question what can be done to offset death in this latter group. Cover and protection can be supplied and indeed used only to a limited extent. Fire bombs must be extinguished, and auxiliary services must be maintained, and parachutists and landing attempts opposed. The navy must, at great risk, expose itself to air attack of unheard of ferocity. The incidence of death and wounding from bombing and machine-gunning and our own shrapnel and anti-aircraft guns will be enormous. Wounds of the chest and deaths from bomb blasts will be greater than ever before in the history of mankind. What can we as thoracic surgeons do to offset such deaths? Relatively little I fear. Since early in 1917 I have been firmly convinced that more armour should be employed to protect those exposed to such attack. The type of helmet employed by the Germans should be adopted by the Allies. It affords greater protection. Fire fighters and soldiers in stationary emplacements should be supplied with body armour at least to protect the chest and upper abdomen. The armour can be light enough to

afford a great measure of protection without unduly burdening the wearer with its weight or interfering with his efficiency. Modern alloys, plastics, and even rubber are available and are effective. The Germans employed it to a limited extent even in the last war. It will not be adopted by the Allies unless great pressure is brought to bear on the armament authorities and governments to make them realize that it is eminently practical, urgently required, and of the very greatest importance. An organization such as this could do much to implement this important method of saving life and reducing the incidence of grave wounds.

Special instructional courses in treatment of chest wounds should be inaugurated for all medical officers.

METHODS OF DEALING WITH CHEST WOUNDS

The mortality from chest wounds varies directly with the nature of the fighting, administrative arrangements, early recognition, immediate adequate first aid and priority evacuation to special centers. The most practical methods to control bleeding, avoid shock and mechanical asphyxia in the first aid post must be developed. In the last war 25 to 30 per cent of the persons with chest wounds died on the field and in the first aid post; another 20 per cent died in the main dressing station and casualty clearing station. Of the so-called fatal chest wounds brought by stretcher-bearers to the aid posts and casualty clearing stations, 60 per cent died within the first forty-eight hours before surgery was employed. Surgical measures for those reaching advance operating centers are well known, but it is for those destined to die from the force of the blast, early hemorrhage and mechanical asphyxia that we of this Association must turn all our talent, thought and power of organization. Their fate is in our hands. Death occurs within two minutes to two hours. A few survive ten to twelve hours. Many of these do not have multiple wounds or grave wounds as have many that are sufficiently fit to be moved to aid posts, advanced dressing stations, and casualty clearing stations. If we can tide these cases over for the first two or three hours, over 80 per cent will be finally saved without doubt; if not, 100 per cent die. In this type of mechanical warfare we cannot ensure the security, safety, and protection to the wounded that was afforded by advanced medical and surgical units in the last war. The tank and dive-bomber have destroyed that.

To my mind it is the most vital and urgent duty of the members of this Association immediately to organize themselves to perfect means, measures, and methods of training and education so that of the 100 per cent of necessarily fatal chest wound victims that die within the first forty-eight hours, the 60 per cent that die within the first few minutes to a few hours, can be tided over into the group that can be evacuated to a surgical center, these above all; and then to train

young surgeons to apply to those so fortunate as to have survived the initial blast and wounds, the skill, experience and knowledge acquired by all of you as a result of surgery of the last war, and the astounding development of thoracic surgery since that time, to which so many of you have made such valuable contributions. Our testing time has come. Thoracic surgery will eclipse all other surgery, indeed all other measures for the saving of life in this struggle for survival, if we play our part and apply the knowledge, experience and ingenuity that is the common property of most members of this progressive and relatively young organization. We, as thoracic surgeons have a major role to play, and we must see to it that we do play such a role. Never has there been such an opportunity as now confronts thoracic surgeons. We must not fail, we will not fail.

PRIMARY (?) MELANOMA OF THE LUNG

A Case Report

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IN VIEW of the fact that pigmented tumors of the lung, whether primary or metastatic, are practically unheard of, we felt that this case was of interest enough to be reported.

Mrs. T., a 48-year-old married woman, consulted Dr. A. Schmitt in December, 1940, and complained of pain to the right of the sternum about the fourth space reflected to the axilla, to the back, and occasionally down the arm. She claimed that she felt exhausted at times, and for several months had a productive yellow expectoration but no blood. No dyspnea was present, appetite was good, and sleep normal. The patient was an inveterate smoker. There was no history of malignancy in family.

Past History.—When 17 years old, the patient had difficulty with her left hip and years later had an orthopedic operation. No other diseases.

Menstrual History.—Her menstrual period began when the patient was 13 years of age and has always been painful, the last period occurring when she was 44 years old. She had a cesarean section fourteen years ago, and the boy is living and well.

Physical examination showed a woman walking with a slight limp and negative findings except for an area of diminished breathing on the right side corresponding to the upper part of the right lower lobe and extending forward. There was no change in vocal fremitus and no rales or bronchial breathing present.

X-ray examination in December, 1940, showed an extensive area of infiltration in the mediastinal half of the right chest around the descending bronchi of the right lower lobe. There was no cavitation. The local area of infiltration was considered to be due probably to infection or malignancy. She deferred entering the hospital until March 3, when she was bronchoscoped and a specimen removed for biopsy from the right middle bronchus. One of us (R. C. S.) reported that the tissue showed the presence of a highly malignant tumor, probably a melanoma.

Another x-ray of the chest on March 4, showed that in the postero-anterior direction the area of infiltration in the mediastinal half of the lower right chest had increased in size. The weight of evidence was in favor of this being a primary malignancy.

In view of the pathologic report, Dr. Schmitt and one of us carefully questioned and examined the patient for the possibility of a primary growth elsewhere. Nothing was found, and the only additional history obtained was that in February, 1939, the patient had fractured two ribs anteriorly on the right side but made a quick recovery.

A complete right pneumonectomy was done, March 15, 1941. The approach was through the third, right intercostal space. Two bands were found running from the lateral chest wall to the lung. After their division the lung was gradually freed from the mediastinum, the growth apparently having not involved the lateral mediastinal wall. No hilal glands were seen; the division of the hilal structures was done high up in sections, ligating and transfixing them with strong silk. There was very little bleeding and throughout the operation, which was done in exactly two

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hours, there were no untoward symptoms, the respirations and pulse remaining regular and the blood pressure slowly dropping to 80 systolic. After being put to bed the patient received 500 c.c. of citrated blood and later 1,000 c.c. of 5 per cent glucose solution. The blood pressure gradually rose so that it was 114/84 at 9 P.M. and outside of the fact that she remained asleep there were no untoward symptoms. The following morning there was no change, the temperature, pulse, and respiration remaining normal, but the patient still was in a stupor. She had no cough, there were no signs of hemorrhage or paralysis, and general reflexes were present. By late afternoon the patient was in a deeper coma and there was some thick mucus in the pharynx. This was aspirated, no blood being obtained. The patient died thirty-six hours after the operation without ever regaining consciousness. There were no convulsions, signs of pneumonia, or of cardiac failure up to the end.

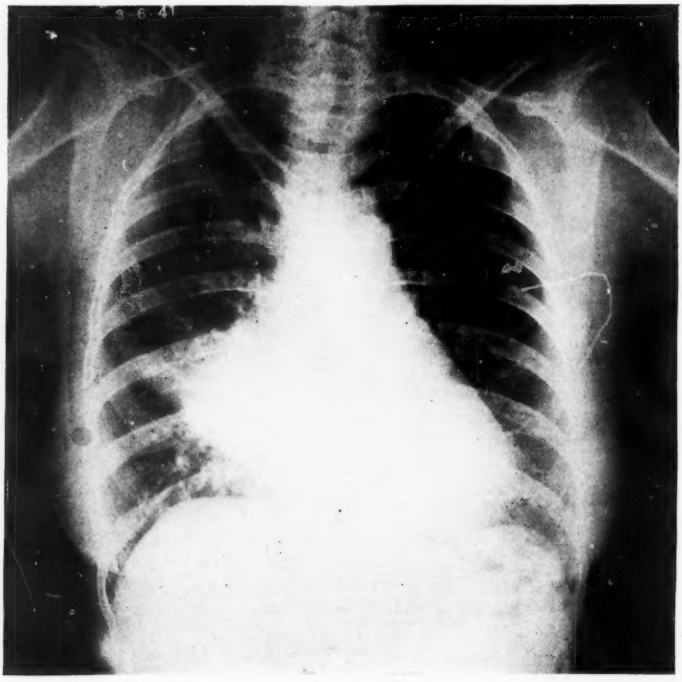

Fig. 1.—X-ray showing antero-posterior view taken March 6, 1941.

Pathologic Report.—

Gross Description: Specimen consisted of an entire right lung weighing 400 Gm., the upper lobe uninvolved. The regions of the middle lobe, the hilus, and the adjoining portion of the lower lobe were infiltrated by a blackish pigmented tumor measuring 5 × 4 × 4 cm. The normal relationships about the hilus were disturbed so that the middle and lower lobes could not be separately identified and the tumor more or less replaced the structures at the hilus, the entire middle lobe, and the adjoining portion of the lower lobe. The bronchus to the upper lobe was free and the bronchi to the lower and middle lobes passed through the tumor. The bronchus to the lower lobe showed tumor tissue in the peripheral portion of the wall but the mucous membrane itself did not appear to have been penetrated. The middle lobe

bronchus was filled with tumor tissue which was only very loosely adherent to the mucous membrane at a few points. It is impossible to say whether the tumor arose in the bronchus primarily or whether it arose outside of the bronchus and penetrated its wall. There were no outlying foci of tumor in the lung. There was more or less atelectasis of the lung tissue adjacent to the tumor and the entire right lung felt fibrosed.

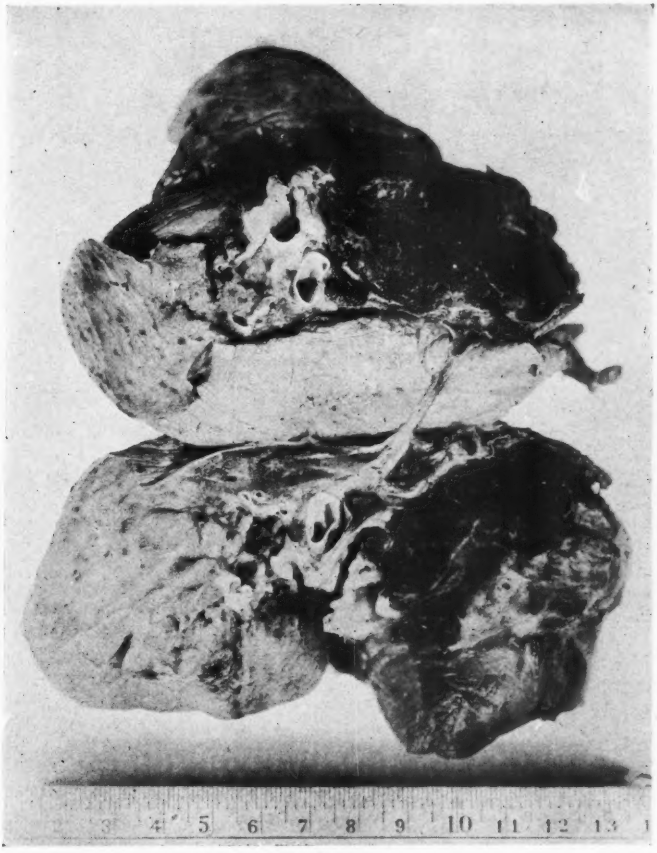

Fig. 2.—Cross-section photograph showing pigmented tumor extending up to the hilum of the lung.

Microscopic Examination: Sections of the tumor showed masses of cells of varying sizes and shapes. Some of these cells were elongated and resembled connective tissue cells. Others were polyhedral and resembled epithelial cells. The cytoplasm was acidophilic. Many of the cells contained blackish brown pigment. The nuclei were extremely variable in size and chromatin content. There was a considerable amount of pigment scattered between the cells. Various iron stains were negative. Scattered through various sections of the tumor were both lung tissue and lymphoid tissue indicating that both structures had been involved. Some portions of the lung tissue showed fibrosis of the interalveolar septa. Sections through the middle lobe

bronchus showed the wall involved including areas where the mucosa had been infiltrated or replaced by tumor tissue. Microscopic and gross study do not allow conclusions to be drawn regarding the origin of the tumor. It may have arisen in the middle lobe bronchus and then spread to the hilar lymph nodes and adjoining lung tissue. It may also have originated as a metastatic lesion in the hilar lymph nodes and spread to the lung and bronchus secondarily.

Diagnosis of melanoma (origin undetermined, possibly primary in bronchus) was made by one of us (R. C. S.).

Fig. 3.—Low power photomicrograph showing pigmented tumor involving the whole lung field.

Unfortunately, it was impossible to obtain an autopsy and, therefore, it could not be determined definitely whether or not this was a primary growth in the lung. The clinical evidence, however, makes one believe that it was. No moles were present in the skin, there was no history of any having been removed, the eyesight and eyegrounds were normal, and there was no clinical evidence of any malignancy anywhere else in the body.

An observation made by Dr. R. Pound, the radiologist, supports this theory. He noted that in the series of films taken of the chest there was

very little appreciable change in the size of the tumor, even after three months, and that after this period of time there was no evidence of other growths in any other part of the lung fields, which is contrary to the usual findings in a metastatic melanoma.

A fairly large number of pigmented tumors are reported in the literature but in the majority the original focus was a mole on the skin or a growth in the eye. After being quiescent for many years they suddenly begin to grow and rapidly metastasize throughout the body, death occurring, as a rule, within a year.

Regarding primary melanotic tumors of the lung, very little is found in the literature, and even the ones reported are questionable because in some instances the autopsies were not complete or the description is not very accurate.

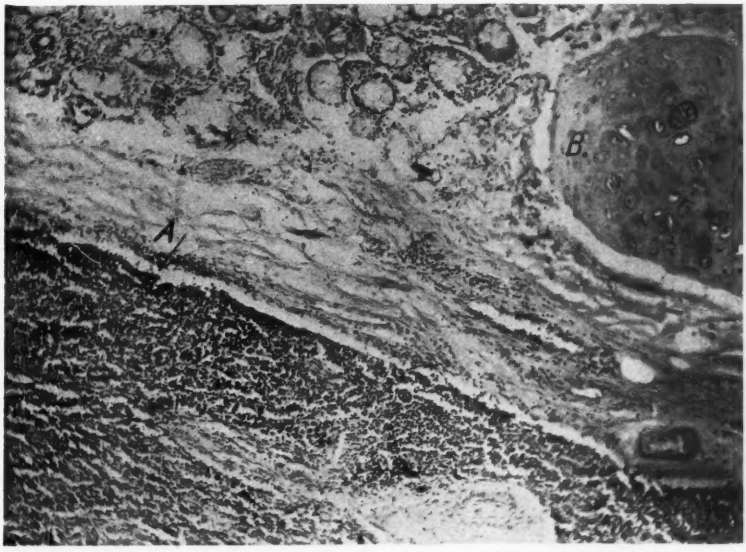

Fig. 4.—Low power photomicrógraph showing tumor extending into bronchus, *A*; *B*, cartilage of bronchus.

Two cases[5] taken from the literature are reported as follows:

CASE 1.—A man, 60 years of age, expectorated large quantities of grumous blood for eight hours. After some medication he improved but three days later he suddenly expired. The autopsy disclosed the presence of two melanotic tumors in the lungs the size of half a hen's egg and the collapsed remains of a third, from which came the black blood. There was extensive pigmentation of the surface of both lungs.

CASE 2.—A man, aged 55 years, who when first seen was breathing with great difficulty. He had cold and clammy skin, a small, rapid pulse, and was expectorating a quantity of frothy serum which had a slight pinkish tinge. Auscultation revealed a dull space in the right lung below the nipple the size of the palm of a small hand. There was no evidence of disease in the heart, kidneys, or elsewhere.

There was no change in the patient's condition for about six months, then he had a sudden hemorrhage of dark grumous blood. This was repeated every few days with very little cough and he finally died of exhaustion.

The autopsy revealed large melanotic tumors of the lower lobe of the right lung, one of which ruptured, causing a peculiar hemorrhage.

Autopsy was limited to the thorax.

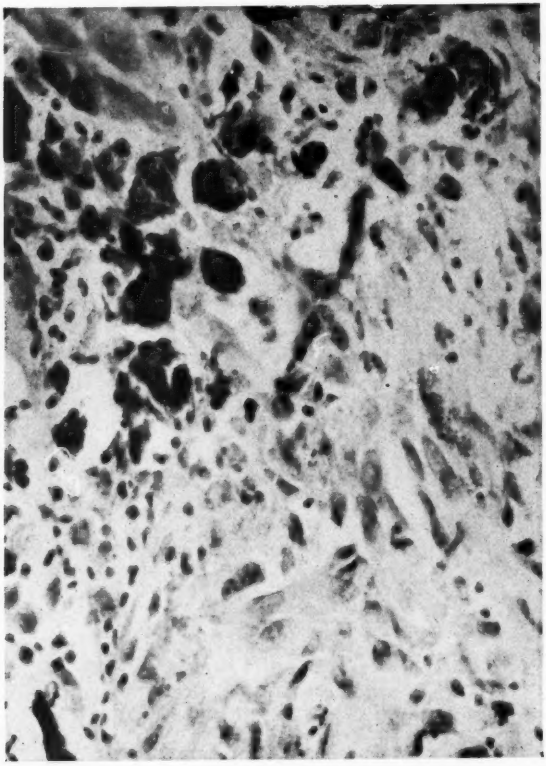

Fig. 5.—High power photomicrograph showing pigmented cells and variety of cells making up the growth.

The following case[4] is another from the literature.

In performing a thoracentesis on an unmarried woman, 40 years of age, force was necessary to insert the trocar, an indication of a thick, leathery pleura. Twenty-four hours later the point of puncture became discolored. In a few days similar discolorations appeared over the left groin and over the lumbar vertebrae. A soft mass developed at this spot and aspiration of the fluid showed the presence of many mitotic cells. The patient rapidly went downhill and died two weeks later.

The autopsy findings showed the left lung to be collapsed. The pleural cavity contained air and about a gallon of coffee-colored fluid. The pleural area was involved at the site where the trocar had been introduced. The mediastinal glands were enlarged to 4 and 6 cm. in diameter.

The primary growth was at the hilum of the right lung were a mass the size of a walnut probably derived from a mediastinal gland. Secondary growth by metastasis involved the lower portion of the upper left lobe and the upper portion of the left lower lobe. The mass in the upper lobe had broken down, perforating the visceral pleura and invading a large area of parietal pleura. Microscopic sections showed melanotic sarcoma with a large predominance of nonpigmented cells.

Comment.—It is difficult to conceive of a primary melanotic tumor originating in the lungs, because normally there are no pigmented cells there. However, both in our case and the case reported by Kunkel and Torrey the evidence is strongly in favor of a primary growth in the chest. There was no evidence of any moles or growths anywhere else, the symptoms were very insignificant for months, and the roentgenograms in our case showed a single tumor and no appearance of any other shadows even three months later, a finding which is contrary to the usual course when metastases develop in the lungs with the original focus elsewhere.

It is interesting to note that the bronchoscopic biopsy revealed the nature of the growth but on examination of the removed lung it could not be determined whether the tumor had originated in the bronchus or the lung tissue proper. At the operation there was no evidence of any involvement of the parietal pleura or of separate pigmented glands at the hilum.

It was unfortunate that an autopsy could not be obtained because of the rarity of this type of tumor in the lung and of the unexplainable death. The patient was in good general condition before the operation, the intratracheal anesthesia was extremely satisfactory, there were no untoward complications throughout the operation, very little blood was lost, the patient was immediately placed in an oxygen tent after the operation, there were no convulsions, the blood pressure remained at a good level, and it was only after twenty-eight hours that signs of cardiac failure developed and she died without ever regaining consciousness.

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USEFUL DEVICES FOR A THORACIC SERVICE

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THE treatment of diseases of the chest includes many unsolved problems and new or developing methods. Any equipment which has been found to facilitate therapy may therefore be of general interest.

The thoracic service at the Wisconsin General Hospital accepts patients, chiefly tuberculous, for special diagnosis and for surgical treatment. There are several hundred procedures a year, including primary and revision thoracoplasties, extrapleural pneumothoraces and plombages, thoracotomies, continuous transthoracic aspirations, oleothoraces, bronchoscopies, occasional lobectomies, as well as whatever extrapulmonary measures are indicated. The use of mechanical aids to collapse therapy (including harnesses, braces, plastic weights) has been previously described.

The devices which are mentioned here have been used during the past four years to fit local situations. They include two adjuncts for transthoracic suction, equipment for a method of postural drainage, an appliance for portable oxygen administration, and a device helpful in aspirations and injections.

I. CONTINUOUS TRANSTHORACIC ASPIRATION

There is a progressive increase in the use of continuous negative-pressure aspiration of chest cavities, both pleural and parenchymal. Obliteration of a pleural space after pneumothorax, and empyema spaces with or without thoracoplasty, is being attempted more frequently, either through intercostal or thoracotomy openings. Deflation of tuberculous cavities (the Eloesser or Monaldi procedure) is a new method of considerable promise, whether alone or after thoracoplasty.

The procedure requires a continuous, stable negative pressure over a period of weeks and a tube which approximates the size of the thoracostomy opening. There are at least three usual disadvantages—leakage from a loose-fitting catheter, local or intercostal pain when it is too tight, and a poorly controlled pressure in the intrathoracic tubing and cavity.

If the tube is passed through sponge rubber, attempts to immobilize it by adhesive tape may cause restriction of mural mobility and further discomfort.

a. An appliance which has seemed to obviate these difficulties has been used in a number of Monaldi and empyema cases. It consists of a solid

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rubber shield about 2 to 3 mm. thick and 6 to 8 cm. in diameter. A drainage tube is selected which fits comfortably into the thoracostomy. A central opening is then punched through the shield, with a diameter about two-thirds that of the tube. The tube is then drawn through the opening the required distance, and the fit is snug without stenosis of the lumen (Fig. 1).

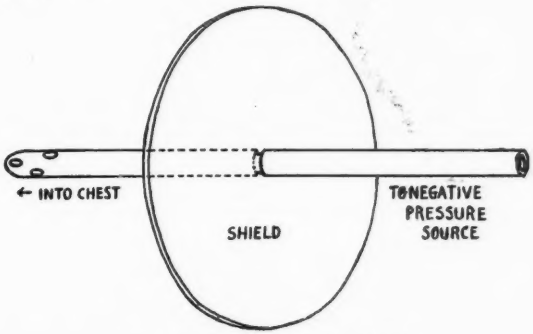

Fig. 1.—Negative pressure shield.

Fig. 2.—Shield and aspiration equipment.

The tube can then be inserted into the chest, the shield lightly cross-hatched to the adjacent skin with adhesive, and the tube marked at the junction with the shield by a thread or a bit of ¼ inch adhesive (Fig. 2). Zinc oxide ointment or tincture of benzoin may be used on the skin beneath the shield. There is no doubt that there is less pain and greater efficiency when the tube is made to fit tightly into the shield rather than the chest wall.

b. The source of the negative pressure may be a bottle arrangement for water displacement, a motor suction pump, or the simple suction jacket (or filter pump aspirator) attached to a water faucet. We have used the latter device, but the hospital water pressure is variable and

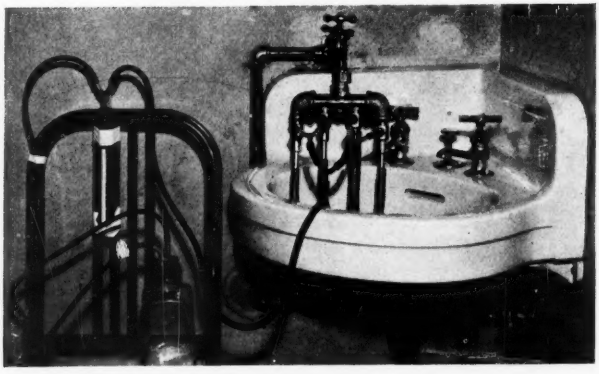

Fig. 3.—Battery of aspirators.

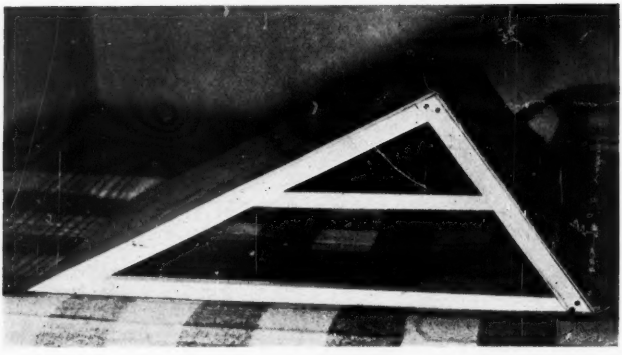

Fig. 4.—Wooden-framed wedge.

Fig. 5.—Bolster wedge.

the pressure to the chest therefore varies even when controlled by a manometer. Dr. Myron Meyers (of this hospital) has found that suction from two faucets produced a stronger, more stable pressure, and on this principle a *battery of aspirators* has been constructed to be used from a single faucet. It works with a markedly improved efficiency (Fig. 3).

II. A METHOD FOR POSTURAL DRAINAGE

The best single method in the treatment of bronchiectasis, and often the only available one, is postural drainage. It may also be of value in cases of lung abscess, and even in tuberculosis.

Recurrent use of a satisfactory posture may be abandoned by the patient because of casual instruction by the physician, a need for help in arranging posture, discomfort during the process, or a weakness which contradicts the use of a precarious position.

A simple method has been in use for two years at the Wisconsin General Hospital. A *wedge-shaped box or bolster,* which can be placed on the bed for use and easily stored away beneath the bed in the intervals, is used. The patient kneels on the bed next to the steeper side, lies forward with his hips bent over the elevated angle, and rests his arms and head upon the bed at the end of the longer side.

The wedge may be constructed of plywood on a light wooden frame, using a sponge-rubber or blanket padding on the surface (Fig. 4). It may also be constructed as a bolster, made of mattress material. We have used the bronchoscopic bolster of Dr. John Jones,* of California, with its similar dimensions and waterproof, washable cover, for this purpose (Fig. 5).

The position is not invariably of value but it is well tolerated, produces a tilt of 20 to 25 degrees, and provides a definite procedure to concentrate the patient's efforts (Fig. 6).

III. A CARRIER FOR PORTABLE OXYGEN ADMINISTRATION

Many patients need oxygen therapy while being transported on a cart from the operating room to the ward or to the fluoroscope. We give all oxygen by means of nasal catheters and, when moving the patients, use a small Waters humidifier and a 20-inch cylinder of the gas. The handling of this equipment, as well as a patient on a cart, was difficult. This prompted the construction of racks made of metal or heavy wire on the undercarriage of the cart (Fig. 7). This has relieved the attendant of the juggling problem and assured the patient of an uninterrupted supply of oxygen.

ADDENDA AND SUMMARY

Other gadgets have been developed for particular uses. The standard ward head-mirrors are fitted with 7 cm. margins of a transparent plastic

*Dr. Jones' data will be published.

Fig. 6.—Use of wedge for postural drainage.

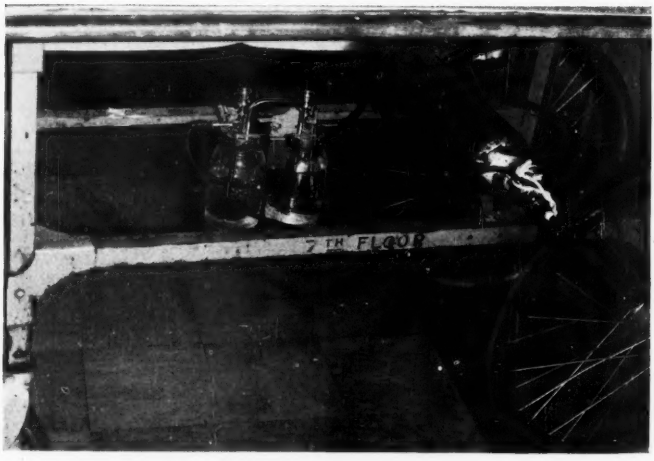

Fig. 7.—Carrier rack for oxygen equipment.

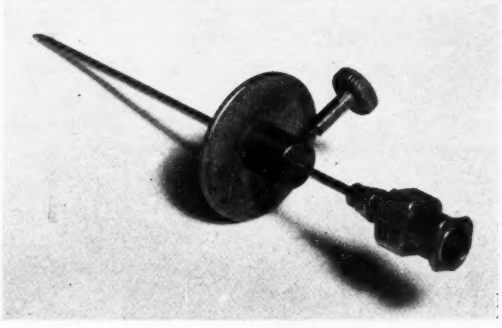

Fig. 8.—Adjustable needle-guard.

to protect the wearer from coughs. A portable table containing all of the equipment for a controlled deflation of spontaneous pneumothorax has been remodelled from a University of Michigan apparatus.

A prime rule in the conduct of aspirations or oleothoraces is to be constantly aware and in control of the position of the needle tip. These procedures should also be as painless, sanitary, and slightly traumatic as possible. A long procedure, with much manipulation, tends to push and pull and waggle the needle in the chest wall. We have used an *adjustable needle guard* which prevents much of the movement and hazard. It consists of a round brass plate the size of a dime, a collar attached, and a long set-screw which runs into the central opening (Fig. 8). It holds the needle at any desired limit. (Pilling makes similar devices for special needles.)

The rubber shield, described above, can be made at the bedside. The other devices have been constructed with the help of the medical school mechanician, the hospital engineer, and the housekeeper.

The literature and personal communications suggest that equipment similar to that described is in use in other clinics. A description of the several together here may increase their usefulness.

EXPERIMENTS IN INTRACARDIAC SURGERY

I. Bacterial Endocarditis

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RECENT advances in thoracic surgery suggest that it is time to take up experimental intracardiac surgery. We are interested in such experimental surgery primarily as it pertains to the treatment of human disease.

No attempt is made here to review the experimental and clinical background of this field. It may suffice to say that the most significant work has been contributed by Cushing,[1] Bernheim,[2] Carrel,[3] Tuffier,[4] Coryllos,[5] Cutler and Beck,[6] Graham and Allen,[7] and Powers.[9]

Recently an opportunity has been provided in this laboratory for the conduct of intracardiac surgical maneuvers which have been directed principally at the production of valvular lesions, intracardiac visualization, and the production and local treatment of bacterial endocarditis.

A SURVEY OF REPORTED METHODS OF EXPERIMENTAL PRODUCTION OF BACTERIAL ENDOCARDITIS

Although not primarily interested in the production of bacterial endocarditis, Rosenbach[10] in 1878, was apparently the first to produce the experimental disease. In this instance, the disease developed in dogs and rabbits on valves that had been injured by passing a rod down the carotid artery. Ribbert[11] in 1885 probably was the first to produce the disease intentionally. His method has, in principle, had repeated recent trials (Dietrich[12]) and was directed at wounding the valves by the injection of staphylococcal suspensions containing starch granules from potato cultures. Dreschfeld,[13] in 1887, was the first to transmit the disease from a human case to an experimental animal.

Since these early efforts a great variety of successful techniques have been presented. These various methods have been conveniently grouped by Blahd, Frank and Saphir[14] into four general types: (1) mechanical procedures directed at leaflet damage with subsequent intravenous introduction of virulent bacteria, (2) simple intravenous injection of bacteria, (3) production of a bacterial focus in the body, and (4) preliminary injection of predisposing substances, with subsequent intravenous injection of virulent organisms.

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From the Surgical Research Laboratories of the Boston City Hospital, Dr. Stephen Maddock, Director.

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The first general method, embracing mechanical injury of the leaflets, followed by spontaneous or induced infection of these lesions has represented the most common as well as the first consistently successful method. The early work of Rosenbach[10] has already been mentioned. The modern controlled counterpart is to be found in the excellent work of Kinsella and Hayes[15] and Kinsella and Meuther.[16] In this last article these workers reported that their animals with damaged leaflets developed bacterial endocarditis after bacterial feedings.

Other ingenious mechanical methods have been sought out by Blahd and his associates[14] and consist of intravenous injections of bacteria in particulate media, such as pulverized carbon,[17] an emulsion of carcinomatous cells,[18] and sterile flour.[19] Finally we must add perhaps the most elaborate procedure of this group, the preliminary fulguration of the mitral valve and subsequent infection of these vulnerable leaflets by the intravenous injection of *Streptococcus viridans*. This last method was devised by Powers.[9] A similar, even more precise technique of fulguration under vision has been devised by Keith.[20]

In principle and practice the first general method has appealed to us as comprehending the most dependable means of producing the experimental disease. It is the method of choice for our purposes also because we are primarily interested in developing a surgical technique that attacks the local lesion rather than the much discussed immunologic, bacteriologic and physiopathologic aspects of the disease.

The second general group, consisting of simple intravenous injection of bacteria, has had abundant trial and inconstant results. It will be recalled that the early and important contributions of Horder[21] and Rosenow[22] were of this type.

Later, Lanfranchi[23] and Fox[24] and still more recently, Cornil, Mosinger and Haimovici,[25] MacNeal and his associates[26] and Lloyd-Jones[27] have used this simple but tedious method with variable results. In general, this approach has been used in work on rabbits, in which instances the disease was produced with ease, and, in those few instances of the injection of dogs, the experimental lesions were produced only with considerable difficulty. Worthy of special comment is the study of Blahd, Frank and Saphir,[14] who used a beta hemolytic streptococcus strain isolated from a spontaneous vegetative endocarditis, discovered by chance, in a dog. This strain reproduced the valvular disease in ten of twenty-five animals into which it was injected.

The third general method aims at the production of bacterial endocarditis by the establishment of a bacterial focus in the body. Most ingenious and most worth while from our point of view has been the work of Friedman, Katz, and Howell,[28] who anchored bakelite bacteria-containing capsules in various places in the heart and great vessels of dogs. To produce the cardiac lesions, the capsules were introduced through the chest wall and myocardium by means of a trocar. This

method is somewhat hazardous to animals and cannot be relied upon for precise localization of the capsule; however, it served the purposes of these investigators admirably. This work should be reviewed by those contemplating studies in this field.

Welch, Murdock, and Ferguson[29] planted *Streptococcus viridans* about the teeth of rabbits, sprayed the rabbits' throats with influenza bacilli, and again produced the disease.

The fourth general method comprehends preparatory injections of various substances such as pitressin as used by Nedzel,[30] or other bacterial strains as demonstrated by Freifeld.[31] Dietrich[32] sensitized rabbits with horse serum, a solution containing casein 5 per cent, colon bacillus and histamine; after sensitization he injected staphylococci and colon bacilli intravenously repeatedly. In his killed animals, all rabbits, he found early lesions.

This general approach to the problem has perhaps the most promising possibilities for immunologists, bacteriologists, and physiologists.[14]

After reviewing the literature, or even the above brief survey, one is impressed by the multiplicity of relationships between rabbits and bacteria that result in endocarditis for the former. Furthermore, one is confronted by the fact that, aside from the pathologic picture, the experimental disease so produced bears little similarity to its human counterpart. To us it appears that these factors tend to invalidate the rabbit as a suitable experimental animal in this work. When one's object is, as ours, to approach the problem from a local surgical point of view, the size of the rabbits' mitral leaflets completes the invalidation.

AN HYPOTHETICAL EXPERIMENTAL ENDOCARDITIS SUITABLE FOR INVESTIGATIONS OF SURGICAL THERAPY

The above-described methods for the production of experimental bacterial endocarditis are of interest in pathologic studies. They also lend to our understanding of the mechanism of origin and perpetuation of this disease. However, they are tedious procedures; they are somewhat uncertain; and the disease as produced in some animals lacks clinical similarity to its human counterpart. These features delay or preclude the phase of investigative work which appears to us to be of prime importance, namely, the treatment of the human disease. In short, the prelude to investigations of therapy in bacterial endocarditis is so expensive in time and animals that few are able to see the program through.

The human lesion is well mapped; it does seem to be the distributing point of the lesions that cause the death, and therefore the destruction or removal of the vegetations or their contained bacteria should cure the sufferer. We are a little discouraged with the possibilities of systemic treatment effecting this. If general or systemic treatment appears to give little promise of success, it then becomes time to turn to local treatment.

Any experimental therapeutic technique directed at the local lesion, that we can now imagine, requires lesions on heart leaflets large enough for surgical approach. The hearts of large dogs are the most practical source available.

Our specifications then resolve themselves into: a fatal disease not too fulminating for therapeutic intervention, that is, the animals should live more than two weeks; this fatal disease should not be so slow in terminating as to render therapeutic assay difficult. The clinical course of the experimental disease should resemble the course of the disease in human beings. The fatal issue must be secondary to leaflet vegetations of bacterial nature. This disease must be produced in animals sufficiently large to permit maneuvers of visualization, excision and/or sterilization. The condition must be technically easy to produce and have a low production mortality.

We present a method of producing an experimental disease that fulfills a large number of the specifications of the suitable hypothetical disease.

EXPERIMENTAL METHOD

Our method, in brief, consists of rendering the free margin of the leaflet vulnerable to vegetation formation by attaching an especially prepared clip. The approach is through the left auricular appendage. In an uncontrolled group of animals under our observation, all of the surviving animals so prepared developed acute bacterial endocarditis presumably due to staphylococci, without any bacterial injection. In a second controlled group, the character of the endocarditis (acute or subacute) and the specific bacterial nature of the vegetations were directed by the administration of sulfanilamide derivatives and subsequent injection of the desired bacterial strains. In this second group there were no operative deaths.

Preoperative Considerations.—Large, young, healthy dogs withstand operation best. These animals have been found to have hearts sufficiently large to render the technical procedure quite simple, although the procedure does lend itself to and has been used in cats. In the large animals any attendant mitral insufficiency is of less consequence. Collie and shepherd dogs have relatively smaller hearts than Airedales, police dogs, hounds and bulldogs, and they also have consistently been found to tolerate surgical procedures less well than the others. It is well to keep animals under observation for a period of from one to two weeks before operation; this provides an opportunity to rule out distemper or other diseases in the animal, to improve the animal's nutritional state and finally, to establish suitable blood levels of the sulfanilamide derivatives, for the controlled disease.

THE OPERATIVE PROCEDURE

Anesthesia.—The intravenous use of nembutal in 6 per cent solution; 1 c.c./5 pounds of body weight. This dosage will provide light anesthesia for well-nourished animals and deep anesthesia for animals in poor general condition. Artificial respiration is essential immediately upon opening the chest. Excellent automatic machines are available, but we have used simply compressed air delivered intermittently through the side arm of a "T" tube attached to an intratracheal tube. Rhythmic pulmonary inflation is then obtained by interrupted positive pressure

delivery. This intermittent positive pressure is produced in the trachea and lungs by alternately covering and uncovering the open end of the "T" tube with a finger. Experience dictates the rate and extent of the inflation. Overinflation causes alveolar rupture. Oxygen can be added to the circuit if desired. Supplementary anesthesia is administered when necessary by injecting ether, 1 to 2 c.c. at a time, into the compressed air tube. If available technicians cannot be depended upon to avoid overinflation of the lungs, a cut-out water valve can be placed in the circuit simply by inserting a "T" tube and placing the end of the side-arm tube the desired distance below the surface of water in a bottle. The distance of the end of the cut-out tube below the water surface then represents the maximum pressure that can be developed in the positive pressure circuit. A reasonable amount of experience with this apparatus convinces one that the simplest possible device is the most satisfactory. Our machine consists of compressed air derived from a wall supply, delivered to the side-arm of a "T" tube fixed on the intratracheal catheter and any available visitor's index finger.

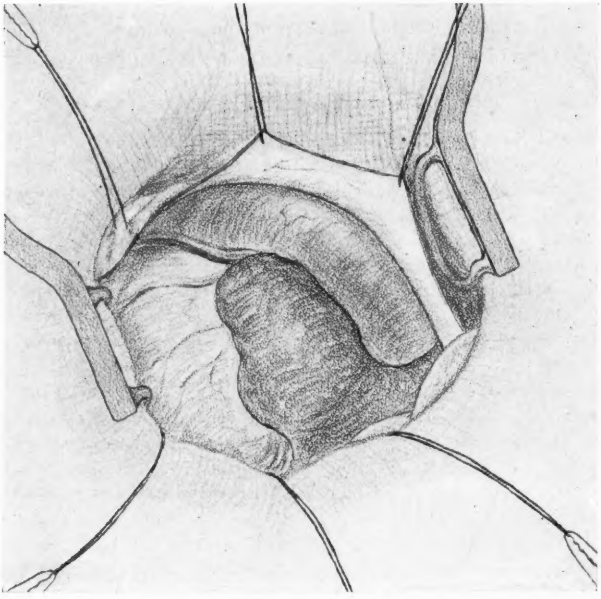


Fig. 1.—The left auricular appendage as seen at operation. The incision has been made in the fifth intercostal space and a self-retaining rib spreader is in place. The pericardium is marsupialized with six silk traction sutures.

The Intratracheal Tube.—The intratracheal catheter is identical with that used in human anesthesia. Ours are discards from the operating room. Balloon cuffs on the intratracheal tube are attractive but are invariably discarded in favor of a moist gauze pharyngeal pack. The ease of insertion, under direct vision, of the intratracheal tube, convinces one of its infinite preference over tracheotomy. The epiglottis is lifted with an Allis clamp giving direct line of vision through the larynx and down the trachea. This makes insertion of the catheter a straightforward maneuver.

The Operation.—The animal is strapped to the table in the dorsal decubitus position with slight right-sided deviation. The chest is shaved, scrubbed with green soap, flushed with alcohol, and painted with Scott's solution.

The drapes are arranged and the incision is carried over the fifth interspace. This interspace is located by counting upward from the thirteenth rib. With remarkable constancy it is found to be one fingerbreadth below the left superior nipple. The incision is carried through the muscles between clamps and/or by retracting in a grid-iron fashion. The intercostal structures are opened down to the parietal pleura for an extent of 2 cm. The parietal pleura is then incised, and, at once, a pneumothorax results and positive pressure artificial respiration must be instituted. The rate and excursion of inflation can now be determined by direct vision. The small incised thoracotomy is extended in the interspace bluntly, by introducing the index fingers and forcibly enlarging the opening. This has the advantage of avoiding hemorrhage from intercostal vessels. Rib spreading, self-retaining retractors are now inserted after covering the wound margins with gauze packs moistened in warm saline (Fig. 1). The pericardium

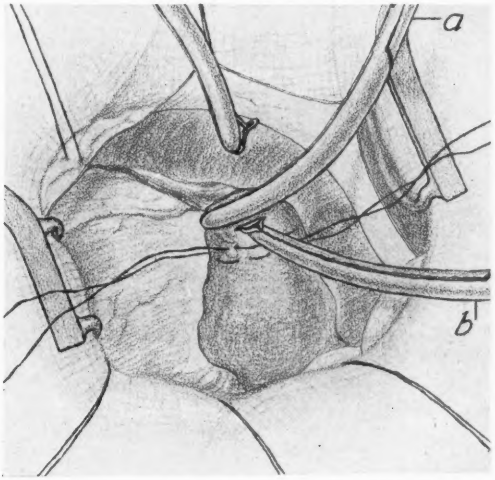


Fig. 2.—The auricular appendage is prepared for the introduction of the clamp carrying the clip. Opposing purse-string sutures are in place. Clamps *a* and *b* close the incision and prevent hemorrhage when approximated end to side as shown.

is picked up a safe distance anterior to the left phrenic nerve; it is incised and the opening is increased bluntly with the index fingers—the purpose again is to prevent bleeding and hematoma formation. The pericardial sac is marsupialized as demonstrated (Fig. 1). This marsupialization helps deliver the heart into a more accessible position and it atraumatically walls out the lungs. Additional control is exerted on the heart by placing clamp *a* on the auricular appendage (Fig. 2). With this as a retractor, the double, opposing purse-string sutures of No. 0 catgut on curved atraumatic intestinal needles are easily placed. This suture must be carried just beneath the epicardium or bleeding will result. Hemostasis is maintained by approximating the point of clamp *b* to the side of clamp *a* when the appendage tip is incised. If the operator is not completely familiar with the mitral orifice, its position can be determined by palpation through the auricle or by inserting an empty closed Kelly clamp through the auricular appendage. The

clip on the clamp is then threaded through the auricular appendage and auricle (Fig. 3), through the mitral ring and past the posterior leaflet—it is then gently pressed against the posterior ventricular wall and gently withdrawn (Fig. 3). The leaflet is always caught by this maneuver, the clip is released, the clamp is withdrawn, and the appendage is closed by the purse strings. This procedure should not be attended by the loss of more than 10 to 20 c.c. of blood. Any blood

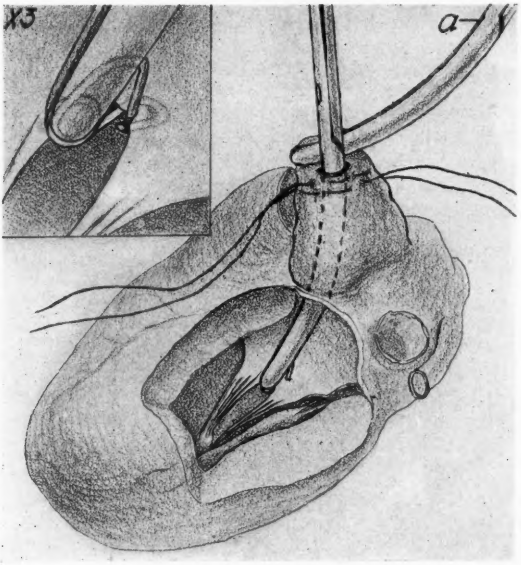


Fig. 3.—Diagrammatic representation of the clip being threaded over the free margin of the posterior mitral leaflet. Inset shows the mechanism by which the clip holds its position.

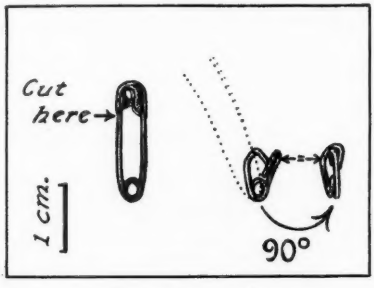


Fig. 4.—Detail of the clip.

should, however, be flushed out of the pericardial sac for it irritates the heart. The upper two-thirds of the pericardium is then closed by tying the silk marsupializing sutures. The lower portion of the pericardium is left open to obviate the danger of tamponade in the event of a pericardial effusion.

The ribs are brought together by pericostal linen sutures, and the incised soft tissues are closed with interrupted silk sutures. Before closing the skin a clamp

is introduced through the closed thoracotomy incision and the lungs are inflated; This is to expel any pneumothorax. The skin is then closed with a subcuticular silk suture, and a sterile dressing is held in place with rubber cement.

If the animal does not take over its own respiration spontaneously after a period of thirty seconds of apnea, stimulation may be provided by a brisk slap on the abdomen or rectal dilatation.

ADDITIONAL COMMENTS ON TECHNIQUE

A word may be added about the preparation of the clips. These are made from small size safety-pins by cutting off the point and fastener. The arms are then bent toward and at right angles to each other as illustrated (Fig. 4). By grasping such a clip in a Kelly clamp as demonstrated, no points are exposed and a smooth shelving edge forms a reverse hook against the convex surface of the Kelly blades (Fig. 3).

Practice on isolated hearts readily demonstrates that such a hook, properly passed over a leaflet and withdrawn, always picks up the free margin. We routinely use the posterior leaflet, although originally both leaflets were clipped but too much destruction and insufficiency followed. The constancy of this maneuver in giving proper application of the clip is attested by the fact that in sixteen consecutive experiments of this type, the animals have been autopsied and the clips were in the elected position in all but one instance.

Presumptive evidence that the clip is properly placed can be gained after the operation by auscultation (a loud systolic blow is heard) and by x-ray examination of the heart.

This operative procedure has been used with very few modifications for the production of tricuspid lesions. With simple alterations the approach and exposure have been used in various reconstructive procedures carried out on the mitral orifice and mitral leaflets.

DISCUSSION

The experimental animals that have been subjected to the procedure described here have been divided into two groups. Group I, comprising sixteen animals, represents the period of development and standardization of technique. Group II, made up of eight animals, represents a technical procedure identical to the first except that in this group the spontaneous bacterial invasion was controlled by chemoprophylaxis and the type of bacterial endocarditis obtained was controlled by the injection of desired strains.

It is of some interest to mention that in all twenty-four of the experiments with this technique, there were no deaths during the operation, nor were there any deaths during the first postoperative day. In the first eight operations there were, however, four deaths that were regarded as operative; in the second eight experiments there were two deaths that must be regarded as operative; and in the last eight operations there have been no operative deaths. Details of these deaths are presented below.

Group I.—In this group of sixteen cases, attention was directed at the development of a safe and simple operative technique. Only limited

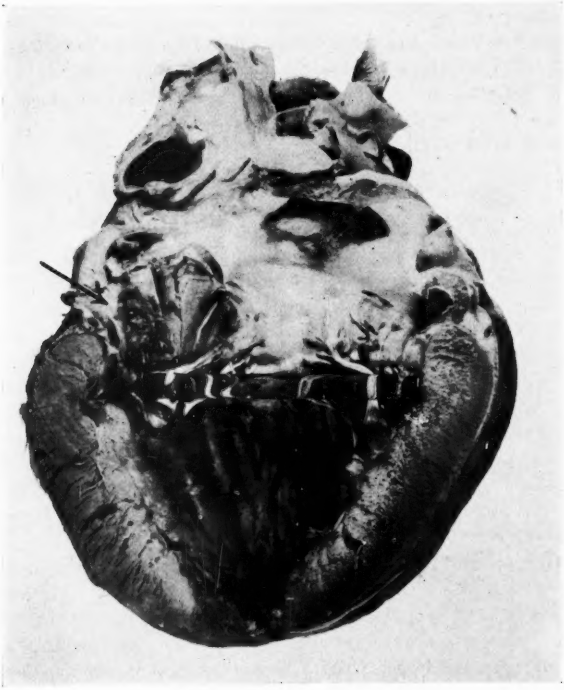


Fig. 5*A*.

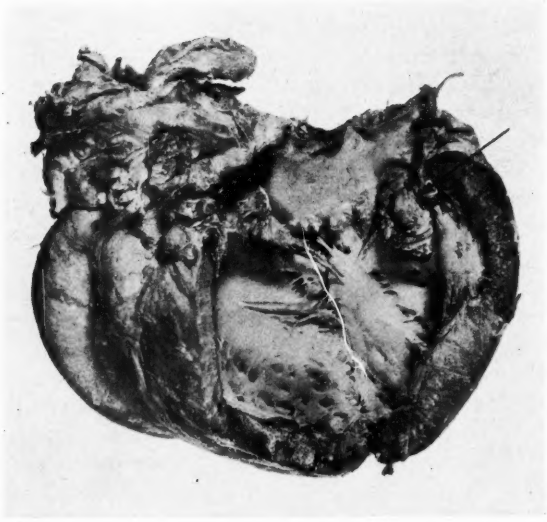


Fig. 5*B*.

Fig. 5*C*.

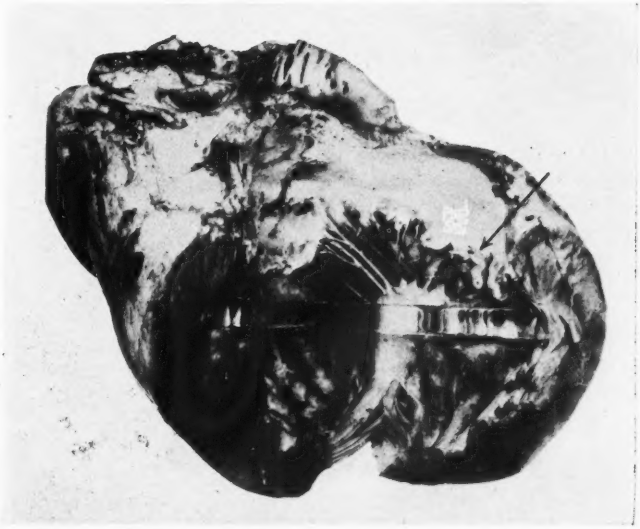


Fig. 5*D*.

Fig. 5.—*A*, *B*, *C*, and *D*, gross specimens demonstrating typical leaflet vegetations formed over the clip.

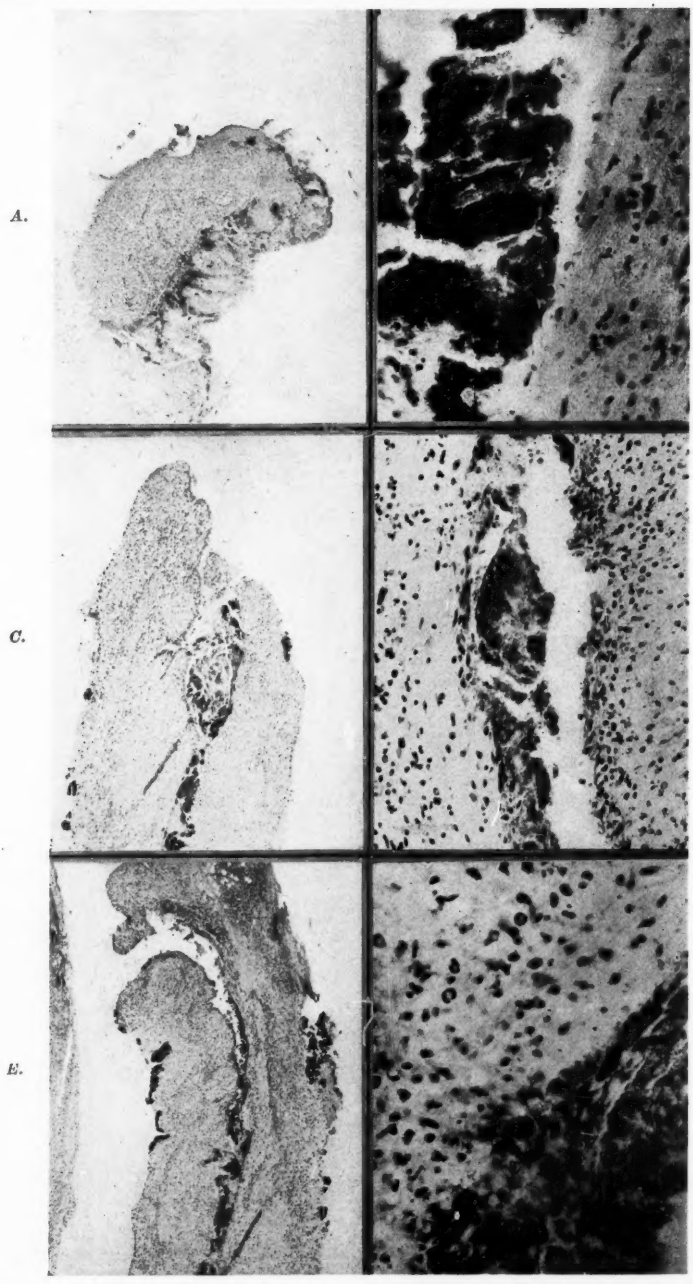
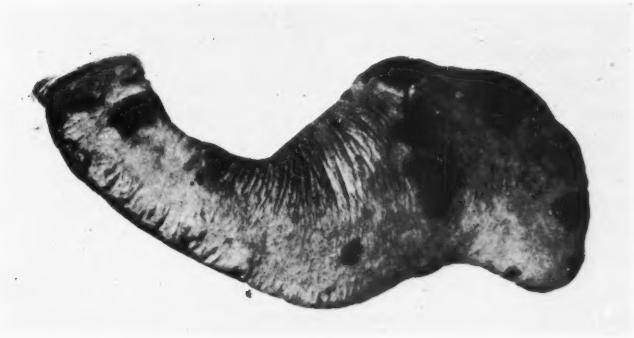
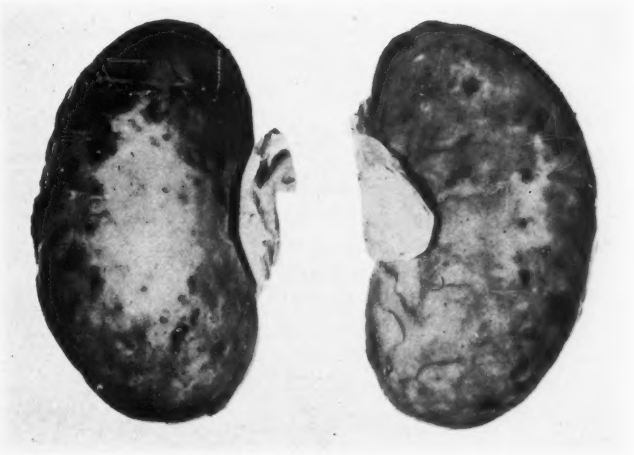


Fig. 6.—Photomicrographs, under high and low power magnification, of typical vegetations on the leaflet margins.

bacteriologic and microscopic studies were carried out. In seven typical, diseased leaflets from which microscopic sections were taken after death —in four animals that had adequate blood culture studies during life and in three direct vegetation cultures at autopsy—the responsible organism was uniformly the staphylococcus. It will be borne in mind that this acute bacterial endocarditis, so closely simulating the gross and



A.



B.

Fig. 7.—Photographs of a spleen (*A*) and kidneys (*B*) studded with septic infarcts. This is a typical manifestation and illustrates the extensive embolic activity.

microscopic appearance of the human disease, developed spontaneously after the clips had been placed on the leaflets. (Figs. 5 and 6.)

Of these sixteen animals, six died within the first two postoperative weeks. Four died with overwhelming infection between the forty-eighth and fifty-sixth postoperative hours. Two deaths occurred during the

second week and were due to pulmonary edema incident to excessive mitral leaflet destruction. In both of these there were well-formed bacterial vegetations on the mitral leaflets. The remaining ten animals ran courses typical of acute bacterial endocarditis with temperatures rising to 103°, and 104° F. and terminating in death within two to twelve weeks. The embolic phenomena were well demonstrated by septic infarcts in the spleen and kidneys (Fig. 7), and these embolic phenomena were responsible for sudden death in at least three instances, two cerebral emboli and one mesenteric embolus.

Group II.—This group, now under investigation, is made up of eight experimental animals, and is set apart as distinct from Group I because the original animals were allowed to develop spontaneous infection of the injured leaflets, whereas this series has been placed on sulfanilamide derivatives for a suitable period before the operation to obtain a protective level. This level is maintained during the day of operation and the first postoperative day when the animals do not eat, by parenteral administration of sodium sulfapyridine. Again, oral administration is instituted once the animals begin to eat. In this way, spontaneous infection seems to be avoided and the acute disease is transmitted by discontinuing the chemoprophylaxis and administering intravenously, doses of 10,000,000 to 15,000,000 pneumococci type I. The subacute disease is similarly instituted by the injection of *Streptococcus viridans* from a case of subacute bacterial endocarditis that is being treated in this hospital.

This group illustrates the low operative mortality that can be expected in the controlled cases. There have been no deaths.

It should be added that this absence of mortality persists in spite of the fact that three of these animals were subjected to coincident cardioscopic examination of the chambers of the left side of the heart at the time of operation. It was possible to visualize the chambers, leaflets, clips in place, and chordae tendinae with moderate facility. The cardioscopes will be described in another publication.

SUMMARY

1. Brief introductory consideration has been given the general field of intracardiac surgery. Pioneer work has been cited.

2. Disappointment has been registered with the present therapy in acute and subacute bacterial endocarditis.

3. In view of the failure to date of generalized systemic attacks on bacterial endocarditis, the possibility of a local approach involving excision, and/or sterilization, with or without intracardiac visualization, has been mentioned.

4. Experimental methods for the production of bacterial endocarditis have been collected and reviewed from the medical literature.

5. An hypothetical experimental bacterial endocarditis suitable for surgical investigations has been outlined.

6. A simple, dependable method for producing bacterial endocarditis of various types has been described.

The writer wishes to express his appreciation to Dr. Stephen Maddock, Director of the Research Laboratories of the Boston City Hospital and to Dr. Stanley J. G. Nowak, Assistant Professor of Surgery of the Harvard Medical School, for their cooperation and suggestions in the conduct of this work. The writer is indebted to Miss Evelyn M. Glidden for the illustrations.

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*The remarkable instrument conceived and used by Dr. Keith was manufactured by the American Cystoscope Company of New York, N. Y., under the supervision of Mr. Fredrick Wappler, President. We have had the privilege of using this instrument in various experiments.

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INDEX TO VOLUME 11

A

Abbott, Osler A. (with Carter, B. Noland, and Hanlon, C. Rollins). Experimental study of tubes made from greater curvature of stomach, 494

Ackman, Douglas (with Vineberg, Arthur M., and Aronovitch, Michael). Thoracoplasty in bilateral pulmonary tuberculosis, 95

Adams, Ralph (with Dufault, Paul). Surgery in pulmonary tuberculosis; study of collapse therapy, with special reference to thoracoplasty, based upon results of thirteen years, 43

Alexander, John. Discussion of surgical and pulmonary tuberculosis, 103
discussion of treatment of endobronchial tuberculosis, 324
(with Sommer, George N. J., and Ehler, Adrian A.). Effect of thoracoplasty upon pulmonary tuberculosis complicated by stenotic tuberculous bronchitis; incidental remarks on pneumonectomy and lobectomy, 308

Allbritten, Frank F., Jr. (with Flick, John B., and Gibbon, John H., Jr.). Use of sulfanilamide in partial and total resection of lung, 187

Allen, Irene (with Skinner, George F., and MacPherson, Lachlan). Thoracoplasty for tuberculosis, 54

American Association for Thoracic Surgery
list of members, 235
list of officers, 234
Presidential Address (Gurd), 1
Rose Lampert Graff Prize, 234, 468
twenty-fifth annual meeting, 234, 468

Anesthesia, respiratory obstruction from bronchiolar constriction during cyclopropane (Rovenstine and Phelps), 565

Anesthesias, with special consideration of possible applications to transpleural surgery; major changes in fundamental relationships of respiratory drive mechanisms during evipal and pentothal (Moyer), 131

Aronovitch, Michael (with Vineberg, Arthur, M., and Ackman, Douglas). Thoracoplasty in bilateral pulmonary tuberculosis, 95

Atelectasis and bronchiectasis (Tannenberg and Pinner), 571

Auerbach, Oscar. Anatomic changes in lungs following thoracoplasty; study of 134 autopsy cases, 21

Aufses, Arthur H. Results in ninety consecutive thoracoplasties for pulmonary tuberculosis, 98

B

Bailey, Charles P. Extraperiosteal pneumonolysis in pulmonary tuberculosis, 326

Berry, Frank B. Discussion of pulmonary function after pneumonectomy in children, 551

Bigger, I. A. Discussion of chronic nontuberculous empyema, 183
discussion of esophagus symposium, 514

Blades, Brian (with Kent, Edward M.). Experimental observations upon certain intracranial complications of particular interest to the thoracic surgeon, 434

Bogen, Emil. Discussion of rest and collapse in pulmonary tuberculosis, 123

Bonilla, J. Lopez, Jr. (with Cid, J. M.). Giant cell tumor of trachea, 210

Brock, William (with Brown, A. Lincoln). Method of treatment of large pulmonary air cysts (balloon cysts) by endocutaneous flap, 617

Bronchiectasis, atelectasis and (Tannenberg and Pinner), 571

Bronchiolar constriction during cyclopropane anesthesia, respiratory obstruction from (Rovenstine and Phelps), 565

Bronchitis, effect of thoracoplasty upon pulmonary tuberculosis complicated by stenotic tuberculous (Alexander, Sommer, and Ehler), 308

Bronchospirography; III. Functional capacity of normal lungs, severely damaged lungs, lungs with strictly parenchymal lesions, thoracoplasty lungs, and re-expanded pneumothorax lungs (Pinner, Leiner, and Zavod), 241
ventilation and oxygen absorption of normal and diseased lungs during nitrogen respiration in the opposite lung (Wright and Woodruff), 278

Bronchus, metastatic carcinoma of lung invading and obstructing (Raine), 216
Brown, A. Lincoln (with Brock, William). Method of treatment of large pulmonary air cysts (balloon cysts) by endocutaneous flap, 617
Burford, Thomas H. (with Graham, Evarts A.). Local use of sulfanilamide in pleural cavity, 203
Burnett, W. E. Discussion of method of treatment of large pulmonary air cysts (balloon cysts) by endocutaneous flap, 630
Butler, Ethan Flagg. Discussion of carcinoma of the lung, 420
discussion of tuberculosis symposium, 321

C

Cadden, A. V. (with Salkin, David). Rest and collapse in pulmonary tuberculosis, 109
new classification of pulmonary tuberculosis, 126
Cancer of lung, with special reference to surgical implications of circumscribed variety, a topographic classification of (Neuhof, Rabin, and Sarot), 388
thoracic esophagus and upper end of stomach (Stephens), 469
Carcinoma, experiences with eight cases of resection of esophagus (Phemister), 484
of lung invading and obstructing bronchus, metastatic (Raine), 216
pneumonectomy for (Harrington), 396
significance of metastasis in primary (Ochsner and DeBakey), 357
resection of esophagus (Wu and Loucks), 516
Carlucci, G. A. (with Schleussner, R. C.). Primary (?) melanoma of lung, 643
Carr, Duane. Discussion of chronic nontuberculous empyema, 184
Carter, B. Noland (with Abbott, Osler A., and Hanlon, C. Rollins). Experimental study of tubes made from greater curvature of stomach, 494
Chamberlain, J. Maxwell (with Gordon, Joseph). Treatment of endobronchial tuberculosis; a review of 100 cases, 292
Cherry, Homer H. Prevention of tissue emphysema following closed pneumonolysis, 451
Chondrosarcoma arising from chest wall and extending into thorax and abdomen, huge (Harper), 446
Churchill, Edward D. Discussion of esophagus symposium, 511
discussion of major changes in fundamental relationships of respiratory drive mechanisms during evipal and pentothal anesthesias, with special consideration of possible applications to transpleural surgery, 150
discussion of pulmonary abscess, 464
discussion of symposium on carcinoma of lung, 419
Cid, J. M. (with Bonilla, J. Lopez, Jr.). Giant cell tumor of trachea, 210
Cole, Dean B. Discussion of method of treatment of large pulmonary air cysts (balloon cysts) by an endocutaneous flap, 634
discussion of surgical and pulmonary tuberculosis, 104
Collapse therapy, with special reference to thoracoplasty based upon the results of thirteen years, a study of (Adams and Dufault), 43
Condon, William B. Regeneration of tracheal and bronchial epithelium, 333
Cournand, Andre (with Lester, Charles W., and Riley, R. L.). Pulmonary function after pneumonectomy in children, 529
Crecca, Anthony D. Discussion of extraperiosteal pneumonolysis in pulmonary tuberculosis, 329
(with Dieffenbach, Richard H.). Analysis of 100 consecutive cases of thoracoplasty with no operative mortality, 65
Crellin, J. Antrim. Discussion of extraperiosteal pneumonolysis in pulmonary tuberculosis, 328
Crimm, Paul D. Laryngoscopic and bronchoscopic ultraviolet lights for treatment of tuberculosis of respiratory tract, 467
Cysts (balloon) large pulmonary air, method of treatment of, by endocutaneous flap (Brown and Brock), 617

D

Davidson, Louis R. Discussion of surgical and pulmonary tuberculosis, 107
Davis, Edgar W. Discussion of method of treatment of large pulmonary air cysts (balloon cysts) by endocutaneous flap, 635
Day, J. C. (with Tuttle, Wm. M., O'Brien, E. J., and Phillips, F. J.). Tuberculous stenosis of major bronchi, 299
DeBakey, Michael (with Ochsner, Alton). Significance of metastasis in primary carcinoma of lungs, 357

Decker, Harry R. Discussion of surgical and pulmonary tuberculosis, 105
Devices for thoracic service, useful (Oatway and Gale), 650
Diaphragmatic hernia with penetrating ulcer of herniated stomach (Frank and Hamilton), 219
Dieffenbach, Richard H. Discussion of chronic nontuberculous empyema, 184
discussion of symposium on surgical and pulmonary tuberculosis, 107
(with Crecca, Anthony D.). Analysis of 100 consecutive cases of thoracoplasty with no operative mortality, 65
Dolley, Frank S. Discussion of treatment of endobronchial tuberculosis and effect of thoracoplasty upon pulmonary tuberculosis complicated by stenotic tuberculous bronchitis, 323
Douglass, Richmond. Discussion of method of treatment of large pulmonary air cysts (balloon cysts) by endocutaneous flap, 636
Dufault, Paul (with Adams, Ralph). Surgery in pulmonary tuberculosis; study of collapse therapy with special reference to thoracoplasty based upon results of thirteen years, 43

E

Eggers, Carl. Discussion of chronic nontuberculous empyema, 183
discussion of esophagus symposium, 509
discussion of pulmonary abscess, 464
Ehler, Adrian A. (with Alexander, John, and Sommer, George N. J.). Effect of thoracoplasty upon pulmonary tuberculosis complicated by stenotic tuberculous bronchitis, 308
Eloesser, Leo. Discussion of effect of thoracoplasty upon pulmonary tuberculosis complicated by stenotic tuberculous bronchitis, 324
discussion of pulmonary abscess, 465
discussion of treatment of endobronchial tuberculosis, 324
Emphysema following closed pneumonolysis, prevention of tissue (Cherry), 451
Empyema, chronic nontuberculous (Singleton and Moore), 169
Endocutaneous flap, a method of treatment of large pulmonary air cysts (balloon cysts) (Brown and Brock), 617
Epithelium, regeneration of tracheal and bronchial (Condon), 333
Esophagus and upper end of stomach, cancer of thoracic (Stephens), 469
for carcinoma, experiences with eight cases of resection of (Phemister), 484
resection of (Wu and Loucks), 516

F

Farber, Jason E. Unexpandable lungs, 424
Finney, George G. Analysis of 104 cases of thoracoplasty for pulmonary tuberculosis, 76
Flick, John B. (with Allbritten, Frank F., Jr., and Gibbon, John H., Jr.). Use of sulfanilamide in partial and total resection of lungs, 187
Frank, L. Wallace (with Hamilton, Joseph E.). Diaphragmatic hernia with penetrating ulcer of herniated stomach, 219

G

Gale, Joseph W. Discussion of extraperiosteal pneumonolysis in pulmonary tuberculosis (Bailey), 329
(with W. H. Oatway, Jr.). Useful devices for a thoracic service, 650
Gibbon, John H., Jr. (with Allbritten, Frank F., Jr., and Flick, John B.). Use of sulfanilamide in partial and total resection of lung, 187
Gordon, Joseph (with Chamberlain, J. Maxwell). Treatment of endobronchial tuberculosis; a review of 100 cases, 292
Graff Prize, 131
Graham, Evarts A. Discussion of carcinoma of lung, 413
discussion of use of sulfanilamide in partial and total resection of lung, 195
(with Burford, Thomas H.). Local use of sulfanilamide in pleural cavity, 203
(with Kent, E. M.). Experimental observations on use of drugs of sulfonamide group in pleural space, 198
Gurd, Fraser B. Discussion of chronic nontuberculous empyema, 182, 185
(Presidential address). Pulmonary abscess; specific method of treatment applicable to both acute and chronic lesions, 1

H

Haight, Cameron. Discussion of a method of treatment of large pulmonary air cysts (balloon cysts) by endocutaneous flap, 630

Hamilton, Joseph E. (with Frank, L. Wallace). Diaphragmatic hernia with penetrating ulcer of herniated stomach, 219

Hanlon, C. Rollins. Discussion of esophagus symposium, 515

(with Carter, B. Noland, and Abbott, Osler A.). Experimental study of tubes made from greater curvature of stomach, 494

Harken, Dwight E. Experiments in intracardiac surgery, I. Bacterial endocarditis, 656

Harper, Fred R. Huge chondrosarcoma arising from chest wall and extending into thorax and abdomen, 336

Harrington, Stuart W. Discussion of symposium on cancer of lung, 423

pneumonectomy for carcinoma of lung, 396

Hayes, John N. Discussion of rest and collapse in pulmonary tuberculosis, 124

I

Intracardiac surgery, experiments in (Harken), 656

Intracranial complications of particular interest to thoracic surgeon, experimental observations upon certain (Kent and Blades), 434

J

Janes, Robert M. Discussion of carcinoma of lung, 421

Jeffers, William A. (with Johnson, Julian, and Margolies, Alexander). Technique of ligation of patent ductus arteriosus, 347

Johns, Frank S. Discussion on symposium on surgical and pulmonary tuberculosis, 105

Johnson, Julian (with Jeffers, Wm. A., and Margolies, Alexander). Technique of ligation of patent ductus arteriosus, 347

K

Kent, Edward M. (with Blades, Brian). Experimental observations upon certain intracranial complications of particular interest to thoracic surgeon, 434

(with Graham, Evarts A.). Experimental observations on use of drugs of sulfonamide group in pleural space, 198

Keys, Ancel (with Paine, John R., and Lynn, David). Observations on effects of prolonged administration of high oxygen concentration to dogs, 151

L

Leiner, George (with Pinner, Max, and Zavod, Wm. A.). Bronchospirography; functional capacity of normal lungs, severely damaged lungs, lungs with strictly parenchymal lesions, thoracoplasty lungs, and re-expanded pneumothorax lungs, 241

Lester, Charles W. (with Cournand, Andre, and Riley, R. L.). Pulmonary function after pneumonectomy in children, 529

Ligation of patent ductus arteriosus, the technique of (Johnson, Jeffers, and Margolies), 347

Lilienthal, Howard. Discussion of chronic nontuberculous empyema, 185

discussion of surgical and pulmonary tuberculosis, 104

Lockwood, Ambrose L. Surgery of chest in war, 637

Loucks, H. H. (with Wu, Y. K.). Resection of esophagus for carcinoma, 516

Lung function, studies of individual (Whitehead, O'Brien, and Tuttle), 266

pneumonectomy for carcinoma of (Harrington), 396

primary melanoma of (Carlucci and Schleussner), 643

use of sulfanilamide in partial and total resection of (Allbritten, Flick, and Gibbon), 187

with special reference to surgical implications of circumscribed variety; a topographic classification of cancer of (Neuhof, Rabin, and Sarot), 388

Lungs following thoracoplasty, anatomic changes in the study of 134 autopsy cases (Auerbach), 21

significance of metastasis in primary carcinoma of (Ochsner and DeBakey), 357

unexpandable (Farber), 424

collapsed by pneumothorax for variable periods of time, a study concerning clinical and anatomic features of re-expanded (Potter), 554

Lynn, David (with Paine, John R., and Keys, Ancel). Observations on effects of prolonged administration of high oxygen concentration to dogs, 151

M

MacPherson, Lachlan (with Skinner, George F., and Allen, Irene). Thoracoplasty for tuberculosis, 54

Maier, Herbert C. Discussion of bronchospirography, 288

Margolies, Alexander (with Johnson, Julian, and Jeffers, Wm. A.). Technique of ligation of patent ductus arteriosus, 347

Meade, Richard H., Jr. Discussion of extraperiosteal pneumonolysis in pulmonary tuberculosis, 330
discussion of use of sulfanilamide in partial and total resection of lung, 196

Melanoma of the lung, primary (?) (Carlucci and Schleussner), 643

Meltzer, Herbert. Bilateral surgical collapse of pulmonary tuberculosis, 227
results of thoracoplasty, 84

Metastasis in primary carcinoma of lungs, significance of metastasis in primary (Ochsner and DeBakey), 357

Meyer, Herbert Willy. Discussion of esophagus symposium, 512

Moore, Richmond L. Discussion of pulmonary abscess, 464

Moore, Robert M. (with Singleton, Albert O.). Chronic nontuberculous empyema, 169

Moyer, C. A. Major changes in fundamental relationships of respiratory drive mechanisms during evipal and pentothal anesthesias, with special consideration of possible applications to transpleural surgery, 131

N

Neuhof, Harold. Discussion of carcinoma of lung, 423
discussion of pulmonary abscess, 463
(with Rabin, Coleman B., and Sarot, Irving A.). Topographic classification of cancer of lung, with special reference to surgical implications of circumscribed variety, 388

O

Oatway, W. H., Jr. (with Gale, J. W.). Useful devices for a thoracic service, 650

O'Brien, E. J. Discussion of bronchospirography and lung function, 289
discussion of pulmonary function after pneumonectomy in children, 551

O'Brien—Cont'd
discussion of symposium on tuberculosis, 321
(with Day, J. C., Tuttle, Wm. M., and Phillips, F. J.). Tuberculous stenosis of major bronchi, 299
(with Whitehead, Walter K., and Tuttle, Wm. M.). Studies of individual lung function; an open circuit procedure with air and low oxygen mixtures as the inspired gases, 266

Ochsner, Alton. Discussion of esophagus symposium, 513
discussion of symposium on cancer of lung, 422
(with DeBakey, Michael). Significance of metastasis in primary carcinoma of lungs, 357

Overholt, Richard H. Discussion on carcinoma of lung, 417
discussion on surgical and pulmonary tuberculosis, 106

Oxygen concentration to dogs, high, observations on effects of prolonged administration (Paine, Lynn, and Keys), 151

P

Paine, John R. (with Lynn, David, and Keys, Ancel). Observations on effects of prolonged administration of high oxygen concentration to dogs, 151

Patent ductus arteriosus, technique of ligation (Johnson, Jeffers, and Margolies), 347

Phelps, McKinnie L. (with Rovenstine, E. A.). Respiratory obstruction from bronchiolar constriction during cyclopropane anesthesia, 565

Phemister, Dallas B. Discussion of esophagus symposium, 515
experiences with eight cases of resection of esophagus for carcinoma, 484

Phillips, F. J. (with Tuttle, Wm. M., O'Brien, E. J., and Day, J. C.). Tuberculous stenosis of major bronchi, 299

Pinner, Max. Discussion of bronchospirography, 288
discussion of rest and collapse in pulmonary tuberculosis, 124
discussion of use of sulfanilamide in partial and total resection of lung, 196
(with Leiner, George, and Zavod, Wm. A.). Bronchospirography; functional capacity of normal lungs, severely damaged lungs, lungs with strictly parenchymal lesions, thoracoplasty lungs, and re-expanded pneumothorax lungs, 241

Pinner—Cont'd
(with Tannenberg, Joseph). Atelectasis and bronchiectasis; an experimental study concerning their relationship, 571
Pleural cavity, local use of sulfanilamide in (Burford and Graham), 203
space, experimental observations on use of drugs of sulfonamide group in (Kent and Graham), 198
Pneumonectomy, experiences with total (Shenstone), 405
for carcinoma of lung (Harrington), 396
in children, pulmonary function after (Lester, Cournand, and Riley), 529
Pneumonolysis in pulmonary tuberculosis, extraperiosteal (Bailey), 326
prevention of tissue emphysema following closed (Cherry), 451
Pneumothorax for variable periods of time, a study concerning clinical and anatomic features of re-expanded lungs which had been collapsed by (Potter), 554
Potter, B. P. Study concerning clinical and anatomic features of re-expanded lungs which had been collapsed by pneumothorax for variable periods of time, 554
Pulmonary abscess (Shaw), 453
specific method of treatment applicable to both acute and chronic lesions (Gurd), 1
air cysts (balloon cysts) by an endocutaneous flap, method of treatment of large (Brown and Brock), 617
function after pneumonectomy in children (Lester, Cournand, and Riley), 529
tuberculosis, analysis of 104 cases of thoracoplasty for (Finney), 76
complicated by stenotic tuberculous bronchitis, effect of thoracoplasty upon (Alexander, Sommer, and Ehler), 308
extraperiosteal pneumonolysis in (Bailey), 326
new classification of (Salkin and Cadden), 126
rest and collapse in (Salkin and Cadden), 109
results in ninety consecutive thoracoplasties for (Aufses), 98
surgery in (Adams and Dufault), 43
thoracoplasty in bilateral (Vineberg, Ackman, and Aronovitch), 95

R

Rabin, Coleman B. (with Neuhof, Harold, and Sarot, Irving A.). A topographic classification of cancer of lungs with special reference to surgical implications of circumscribed variety, 388
Raine, Forrester. Metastatic carcinoma of lung invading and obstructing bronchus, 216
Respiratory drive mechanisms during evipal and pentothal anesthesias, with special consideration of possible applications to transpleural surgery, major changes in the fundamental relationships of (Moyer), 131
Rienhoff, William F., Jr. Discussion of symposium on carcinoma of lung, 415
Riley, R. L. Discussion of pulmonary function after pneumonectomy in children, 553
(with Lester, Charles W., and Cournand, Andre). Pulmonary function after pneumonectomy in children, 529
Rovenstine, E. A. (with Phelps, McKinnie L.). Respiratory obstruction from bronchiolar constriction during cyclopropane anesthesia, 565

S

Salkin, David (with Cadden, A. V.). Rest and collapse in pulmonary tuberculosis, 109
new classification of pulmonary tuberculosis, 126
Sarot, Irving A. (with Neuhof, Harold, and Rabin, Coleman B.). Topographic classification of cancer of lungs with special reference to surgical implications of circumscribed variety, 388
Schleussner, R. C. (with Carlucci, G. A.). Primary (?) melanoma of lung, 643
Shaw, Robert R. Pulmonary abscess, 453
Shenstone, Norman S. Discussion of symposium on cancer of lung, 423
experiences with total pneumonectomy, 405
Singleton, Albert O. (with Moore, Robert M.). Chronic nontuberculous empyema, 169
Skinner, George F. (with MacPherson, Lachlan, and Allen, Irene). Thoracoplasty for tuberculosis, 54

Sommer, George N. J. (with Alexander, John, and Ehler, Adrian A.). Effect of thoracoplasty upon pulmonary tuberculosis complicated by stenotic tuberculous bronchitis, 308

Stephens, H. Brodie. Cancer of thoracic esophagus and upper end of stomach, 469
discussion of esophagus symposium, 514

Stomach, experimental study of tubes made from greater curvature of (Carter, Abbott, and Hanlon), 494
upper end of, and cancer of thoracic esophagus (Stephens), 469

Sulfanilamide in partial and total resection of lung, use of (Allbritten, Flick, and Gibbon), 187
in pleural cavity, local use of (Burford and Graham), 203

Sulfonamide group in pleural space, experimental observations on use of drugs of (Kent and Graham), 198

Surgery, experiments in intracardiac; I. Bacterial endocarditis (Harken), 656
of chest in war (Lockwood), 637

T

Tannenberg, Joseph. Discussion of observations on effects of prolonged administration of high oxygen concentration to dogs (Paine, Lynn, and Keys), 168
(with Pinner, Max). Atelectasis and bronchiectasis; an experimental study concerning their relationship, 571

Thompson, Samuel A. Discussion of bronchospirography, 287

Thoracic service, useful devices for (Oatway, Jr. and Gale), 650

Thoracoplasty, anatomic changes in lungs following (Auerbach), 21
based upon results of thirteen years, study of collapse therapy, with special reference to (Adams and Dufault), 43
for pulmonary tuberculosis, analysis of 104 cases (Finney), 76
for tuberculosis (Skinner, MacPherson, and Allen), 54
in bilateral pulmonary tuberculosis (Vineberg, Ackman, and Aronovitch), 95
results of (Meltzer), 84
upon pulmonary tuberculosis complicated by stenotic tuberculous bronchitis, effect of (Alexander, Sommer, and Ehler), 308
with no operative mortality, analysis of 100 consecutive cases of (Dieffenbach and Creech), 65

Thoracoplasties for pulmonary tuberculosis, results in ninety consecutive (Aufses), 98

Trachea, giant cell tumor of (Cid and Bonilla), 211

Transpleural surgery, major changes in fundamental relationships of respiratory drive mechanisms during evipal and pentothal anesthesias, with special consideration of possible applications to (Moyer), 131

Tuberculosis, endobronchial, treatment of (Chamberlain and Gordon), 292
of the respiratory tract, laryngoscopic and bronchoscopic ultraviolet lights for treatment of (Crimm), 467
pulmonary, analysis of 104 cases of thoracoplasty for (Finney), 76
a new classification of (Salkin and Cadden), 126
bilateral surgical collapse of (Meltzer), 227
rest and collapse in (Salkin and Cadden), 109
results in ninety consecutive thoracoplasties for (Aufses), 98
surgery in (Adams and Dufault), 43
thoracoplasty for (Skinner, MacPherson, and Allen), 54
in bilateral pulmonary (Vineberg, Ackman, and Aronovitch), 95

Tubes made from greater curvature of stomach, an experimental study of (Carter, Abbott, and Hanlon), 494

Tuttle, William M. Discussion of extraperiosteal pneumonolysis in pulmonary tuberculosis, 328
(with O'Brien, E. J., Day, J. C., and Phillips, F. J.). Tuberculous stenosis of major bronchi, 299
(with Whitehead, Walter K., and O'Brien, E. J.). Studies of individual lung function; an open circuit procedure with air and low oxygen mixtures as the inspired gases, 266

V

Vineberg, Arthur M. (with Ackman, Douglas, and Aronovitch, Michael). Thoracoplasty in bilateral pulmonary tuberculosis, 95

W

War, surgery of chest (Lockwood), 637

Whitehead, Walter K. Discussion of bronchospirography, 290

(with O'Brien, E. J., and Tuttle, Wm. M.). Studies of individual lung function; an open circuit procedure with air and low oxygen mixtures as the inspired gases, 266

Woodruff, Warriner (with Wright, George W.). Bronchospirography; ventilation and oxygen absorption of normal and diseased lungs during nitrogen respiration in opposite lung, 278

Wright, George W. (with Woodruff, Warriner). Bronchospirography; ventilation and oxygen absorption of normal and diseased lungs during nitrogen respiration in opposite lung, 278

Wu, Y. K. (with Loucks, H. H.). Resection of esophagus for carcinoma, 516

Z

Zavod, Wm. A. (with Pinner, Max, and Leiner, George). Bronchospirography; functional capacity of normal lungs, severely damaged lungs, lungs with strictly parenchymal lesions, thoracoplasty lungs, and re-expanded pneumothorax lungs, 241